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DISSERTATION

**THE EFFECT OF MILD MOTION SICKNESS
AND SOPITE SYNDROME ON
MULTITASKING COGNITIVE PERFORMANCE**

by

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March 2013

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**THE EFFECT OF MILD MOTION SICKNESS AND SOPITE SYNDROME ON
MULTITASKING COGNITIVE PERFORMANCE**

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ABSTRACT

This research investigated the effects of mild motion sickness and sopite syndrome on multitasking cognitive performance. Fifty-one healthy individuals (45 males, 6 females) were recruited in three data collection periods from the pool of Naval Postgraduate School (NPS) students, faculty, and staff. Participants from the 2010 and 2011 data collection periods were randomly assigned to one of two groups, M-NM (n=20, motion in the first session, no motion in the second) or NM-M (n=19, no motion in the first session, motion in the second). All participants (n=12) from the 2012 data collection were assigned to group “NM-NM” and did not experience motion in either session.

On average, reported severity of motion sickness was mild. In this study, cognitive multitasking performance deteriorated with the development of mild motion sickness; however, this result was confounded by an order effect. Performance differences between Symptomatic and Asymptomatic participants in composite (9.43%), memory (31.7%), and arithmetic (14.7%) task scores were significant only in the second experimental session. Furthermore, results suggest that performance retention between sessions in a novel cognitive multitasking environment is not affected by mild motion sickness. We postulate that the differential effect of session on the association between symptomatology and multitasking performance may be related to the attentional resources allocated to performing the multi-task. Results suggest an inverse relationship between motion sickness effects on performance and the cognitive effort focused on performing a task. Lastly, a revised definition of sopite syndrome is proposed, addressing the limitations of earlier approaches.

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LIST OF ACRONYMS AND ABBREVIATIONS

CPM	Cycles per Minute
ECG	Electrocardiography
EDA	Electrodermal Activity
EGG	Electrogastrography
HFR	Human Factors Research, Inc.
HMD	Head Mounted Display
Hz	Hertz
MSAQ	Motion Sickness Assessment Questionnaire
MSI	Motion Sickness Incidence
MSSQ	Motion Sickness Susceptibility Questionnaire
NPS	Naval Postgraduate School
RPM	Rounds Per Minute
SAS	Supervisory Attentional System
SEM	Standard Error of the Mean
SES	Surface Effect Ship
SSS	Stanford Sleepiness Scale

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EXECUTIVE SUMMARY

The purpose of this research was to investigate the effects of mild motion sickness and sopite syndrome on multitasking cognitive performance. The experiment was conducted at the Naval Postgraduate School (NPS) in three data collection periods. The first period was January through February 2010. The second period occurred a year later. The third period was May through June 2012. Participants included 51 healthy, non-smoking individuals (45 males, 6 females) recruited from the pool of NPS students, faculty, and staff. Participants from the 2010 and 2011 data collection periods were randomly assigned to one of two groups, M-NM (n=20, motion in the first session, no motion in the second) or NM-M (n=19, no motion in the first session, motion in the second). All participants (n=12) from the 2012 data collection were assigned to group “NM-NM” and did not experience motion in either session. All participants had normal vision and hearing and were screened before the beginning and during the study for illnesses or other issues that could affect their test performance. The nauseogenic motion stimulus included the superposition of three independent 0.167 Hz sinusoidal motions. The z-axis had a +/- 2 inches displacement (heave), while the y and x axes had a +/- 15 degrees roll and pitch. In general, the average severity of motion sickness in our study was mild.

Results indicated that cognitive multitasking performance deteriorated with the development of mild motion sickness, confounded by an order effect. Only during the second experimental session motion sickness and soporific symptoms had a pronounced negative association with performance. Significant performance differences were identified between Symptomatic and Asymptomatic participants in composite (9.43%), memory (31.7%), and arithmetic (14.7%) task scores in the second experimental session but not the first.

We postulate that the differential effect of session in the association between symptomatology and multitasking performance may be related to the attentional resources allocated to performing the multi-task. This hypothesis is based on the fact that the two experimental sessions differed only in the degree of skill the participants

acquired. Results suggest an inverse relationship between motion sickness effects on performance and the cognitive effort focused on performing a task.

Furthermore, results suggest that performance retention between sessions in a novel cognitive multitasking environment is not affected by mild motion sickness. Lastly, a revised definition of sopite syndrome is proposed, addressing the limitations of earlier approaches.

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Lastly, this work is dedicated to my parents, Panagiota Pavlopoulou and George Matsangas. Now that this effort is coming to its end, I remember my father citing Napoleon Bonaparte: *“Every hour of lost time is a chance for future misfortune.”*

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I. INTRODUCTION

A. DISSERTATION STATEMENT

Motion sickness is a physiological response to a specific frequency and acceleration space of provocative motion (McCauley, Royal, Wylie, O'Hanlon, & Mackie, 1976; Money, 1970; O'Hanlon & McCauley, 1974). Although motion sickness is not rare, it is usually infrequent for a given individual. The occurrence of motion sickness depends on the severity of the motion, the susceptibility of the individual, and their recent experience in motion environments. Attention is paid to motion sickness mainly when the severe symptoms are apparent, that is, when people vomit or show symptoms of extreme malaise. The operational significance of motion sickness to the military has its roots in the second World War era (Reason & Brand, 1975, p. 31; Wendt, 1944; Zwerling, 1947). Naval personnel often perceive motion sickness as an important issue only when performance deteriorates. Reduced performance becomes obvious when motion sickness interferes with the assigned tasks or duties. The reduced performance criterion, however, is fuzzy and not easily determined.

Our research focuses on this grey area of motion sickness, where some symptoms exist, but the severity of malaise is low. As described later, the term motion sickness encapsulates a constellation of symptoms, ranging from headache to emesis. Among these symptoms, emesis is unique because it is an objectively observable behavioral marker and is binary; that is, it occurs or it does not occur. The rest of the symptoms, though, depict a range of severity levels. In general, early motion sickness research focused on severe cases of emesis or nausea where individuals were incapacitated and could not continue to perform. Nevertheless, Wendt (1944) noted that low severity motion sickness is characterized only by emotional depression and loss of interest in work. It was not until the 1970s, that a more systematic approach began to emerge a) regarding a cluster of symptoms associated with drowsiness (sopite syndrome), and b) of the association between non-incapacitating symptoms and performance (Graybiel & Knepton, 1976; Lawson & Mead, 1998).

On the other hand, even nausea and gastric activity demonstrate a wide range of severity from “just noticeable activity” to retching as a prodromal response to vomiting. In this dissertation, the term “mild motion sickness” is used to describe those motion sickness-related symptoms that are not incapacitating, and do not significantly interfere with the individual’s ability to perform. In such mild severity situations, personnel often believe that a well-trained and motivated crew can overcome mild motion sickness effects. Or, to rephrase Davenport (2007, p. 5): “our culture, especially in the military, holds that somehow training, habit, motivation or attitude can overcome...” mild motion sickness.

Therefore, the question arises: is performance affected by mild motion sickness and sopite syndrome?

B. PROBLEM STATEMENT

Numerous studies have approached the problem of performance reduction because of motion sickness, both in the laboratory and in the field (for example Alexander, Cotzin, Hill, Ricciuti, & Wendt, 1945d; Hettinger, Kennedy, & McCauley, 1990). These research efforts conclude that motion sickness affects human task performance in a complicated manner. They suggest that motion sickness affects human performance more when tasks are complex, require long periods of effort or sustained performance, and offer an opportunity for individuals to control the pace of their efforts. Hettinger and colleagues (1990) suggested that performance decrements because of motion sickness are mainly attributed to reductions in subject motivation rather than deterioration in the performance capacity of the sick individuals. Tasks begin to be viewed by the sufferer as nonessential, and are subject to the discretion of the crewman. Other studies have come to the same conclusions: “Motion sickness was generally incapacitating to the Subjects, rendering most incapable of performing all but the simplest experimental tasks. Moreover, sick Subjects tended to exercise their prerogative of leaving the test environment, in some cases long before the scheduled completion of their motion exposure” (O’Hanlon, Miller, & Royal, 1977, p. 6).

In general, evidence suggests that human performance can be degraded because of the effects related to the symptomatology referred to as “sopite syndrome,” a group of symptoms related to drowsiness (Graybiel & Knepton, 1976; Lawson & Mead, 1998). There are a number of potential problems associated with sopite syndrome, for example issues related to health and safety, vigilance performance, or driver fatigue (Brill, Hancock, & Gilson, 2003; Kennedy, Massey, & Lilienthal, 1995; Lawson & Mead, 1998). The severity and occurrence of drowsiness, as a symptom related to sopite syndrome, is dominant, exceeding by far other symptoms of motion sickness (Bos, Damala, Lewis, Ganguly, & Turan, 2007; McCauley, Pierce, & Matsangas, 2007).

Nevertheless, the system design community, which determines the extent of motion allowed on a given platform, still relies on standards based on the Human Factors Research, Inc. (HFR) experiments that predict the percent of people who will vomit (McCauley et al., 1976; O’Hanlon & McCauley, 1974). The reason for relying on emesis may be found in the fact that existing research hasn’t provided the appropriate level of solid, quantified, conclusions regarding performance deterioration associated with sopite syndrome, or mild motion sickness severity or symptoms excluding vomiting (Matsangas, McCauley, & Papoulias, 2009, 2010).

The present research focuses on expanding the understanding of the the relationship between mild motion sickness, soporific effects, and cognitive multitasking performance. This experiment used a relatively mild real motion stressor; assessment was based on a multitasking cognitive work environment, using subjective (self-report), and objective (psychophysiological) metrics of motion sickness severity.

C. CONTRIBUTIONS

The aim of this study is to fill the gap in our knowledge regarding the indirect effects of motion (Colwell, 2005). More specifically, we focus on the effects of soporific symptoms and mild motion sickness on cognitive multitasking performance. The existing knowledge regarding the quantifiable effect of sopite syndrome on performance is not sufficient to inform optimal naval architecture and related motion-based system designs.

From an operational perspective, this research may contribute to:

- quantifying the effects of mild motion sickness and sopite syndrome on cognitive multitasking performance.
- increasing the validity of military simulations concerning human-in-the-loop performance.

D. HYPOTHESIS

This dissertation will assess the hypothesis that multitasking cognitive performance is significantly reduced by mild motion sickness and soporific effects.

E. DISSERTATION ORGANIZATION

This dissertation is organized into the following chapters:

Chapter II: Background and Related Work. This chapter covers the domains that help develop the experimental objectives: motion sickness symptomatology, sopite syndrome, and the effect of these two on cognitive performance. In addition, the background covers the area of motion sickness measurement and detection tools.

Chapter III: Methods. This chapter covers the methodology and demographic data of the participants used in the experiment. It includes a description of the rationale leading to the experimental design.

Chapter IV: Analysis and Results. This chapter covers the analysis and the corresponding results to address the study objectives.

Chapter V: Discussion. This chapter describes briefly the results of the study, and discusses the findings in relation to existing research.

Chapter V: Recommendations. This chapter builds upon the findings of the study, and provides ideas for future explorations.

II. BACKGROUND AND RELATED WORK

A. MOTION SICKNESS

Motion sickness in healthy individuals is a common syndrome that occurs at the presence of either real or apparent motion. Motion sickness is a general term describing a number of symptoms, ranging from discomfort to emesis, related to the existence of real or apparent motion. Depending on the situation in which the symptoms occur, there are a number of other names related to motion sickness including nausea, cybersickness, space adaptation syndrome, simulator sickness, kinetosis, and motion adaptation syndrome. Benson (1988) defined motion sickness as a condition that occurs when people (as well as fish and other animals) are exposed to real or apparent motion stimuli with which they are unfamiliar and to which they are unadapted. The term “motion sickness” is a misnomer (Benson, 1999) because a) motion sickness may be induced in the absence of motion such as during a virtual reality simulation, and b) “sickness” implies that it is a type of disease, when in fact it is a perfectly normal response of a healthy individual without any functional disorder. In fact, only people with a non-functional vestibular system seem to be immune to motion sickness (Kennedy, Graybiel, McDonough, & Beckwith, 1968).

There is a wide range of susceptibility to motion sickness, both among people because of individual differences, and within an individual on different occasions (Dobie, 2000; Griffin, 1990; Kennedy, Dunlap, & Fowlkes, 1990). Susceptibility to motion sickness has been linked to several factors. Compared to males, females of the same age are less susceptible to motion sickness (Benson, 1999; Jokerst et al., 1999; Lawther & Griffin, 1988; Lentz & Collins, 1976). Susceptibility changes with age, with aging adults becoming more less sensitive (Benson, 1999, 2002; Lawther & Griffin, 1988; Wertheim, 1998). A person’s personality (increased susceptibility is associated with increased neuroticism), as well as past sensory experiences are known factors (Collins & Lentz, 1977; Gordon et al., 1994; Kottenhoff & Lindahl, 1960; Reason, 1972; Steele, 1961). Furthermore, sleep deprivation is also known to interfere with the vestibular system habituation process (Dowd, 1974).

Numerous studies have dealt with the incidence of motion sickness at sea, but the results are extremely variable. For example, Hill (1936) noted an incidence of over 90%, Chinn (1951) reported a 25% to 30% incidence, whereas Pethybridge (1982) reported that 10% to 30% of participants experienced motion sickness. Studies on small marine craft have found an incidence of emesis ranging from 11 to 70% of the crew depending on the sea state (Holling, McArdle, & Trotter, 1944; Llano, 1955; Tyler & Bard, 1949). Emesis was experienced by 15 to 60% of the passengers aboard ships making winter crossings of the Atlantic Ocean during the first few days of the crossing (Chinn, 1956). However, laboratory experiments may underestimate motion sickness incidence compared to real life data (for example, Goto & Kanda, 1977). Some researchers attributed this difference to the fact that laboratory research, generally, focuses on simple sinusoidal motion whereas in real-life moving platforms, individuals face complex periodic waveforms and aperiodic motions (Guignard & McCauley, 1982).

Reason and Brand (1975) explain susceptibility to motion sickness by noting that the body continuously expects to receive signals from its sensory organs in a recognizable pattern. Thus, motion sickness is the normal response during the period where the body is gradually learning a new signal combination which differs from the known pattern (mismatch). In this case, susceptibility to motion sickness can be seen as the rate at which the internal model (of expected motion stimuli) can be changed. This rate is affected by three factors: receptivity, adaptability, and retentiveness. Receptivity is the individual's internal amplification of the motion stimulus. Adaptability is the rate at which the internal model is changed. Retentiveness is the individual's ability to retain the internal model and continuously adapt it to a motion environment in successive exposures (Reason, 1972).

1. Symptomatology, Time Development, and Adaptation

The signs (observable) and symptoms (not observable) of motion sickness include breathing irregularities, yawning, sensation of warmth, disorientation, drowsiness, facial pallor, cold sweating, nausea, and emesis (Benson, 1988; Chinn & Smith, 1955; Clark & Graybiel, 1961; Crampton, 1955; Hemingway, 1944; Reason & Brand, 1975).

Assuming continuation of the motion environment, motion sickness symptomatology demonstrates a progressive development over time. Schwab (1954) noted that motion sickness includes a wide range of minor symptoms that escalate before actual nausea and vomiting occurs.

The sequence of the symptoms is idiosyncratic and depends on individual susceptibility and the intensity of the motion stimuli. Soon after initial exposure to a provocative stimulus, susceptible individuals will develop identifiable symptoms; in some very rare cases, these symptoms may occur after only a few seconds (Graybiel, Deane, & Colehour, 1969). This progressive increase in the severity of symptomatology is eventually followed by adaptation, a decrease in the observed symptoms over time as the individual adapts to the motion environment. Some operational studies have identified a two-day adaptation period (McCauley, Matsangas, & Miller, 2005; Wiker & Pepper, 1981). However, in about 5% of the population, adaptation does not appear to occur (Hemingway, 1945; Tyler & Bard, 1949).

2. Causal Factors of Motion Sickness

The most widely accepted theory regarding the causal factors of motion sickness is named by several names: sensory conflict mismatch theory, sensory rearrangement theory (Reason & Brand, 1975) or neural mismatch theory (Benson, 1999). In general, this theory postulates that the cause of motion sickness is the mismatch between the pattern of information from the spatial senses and that retained in memory storage of previous experience. It is hypothesized that the latter, also called a neural store, is an internal model of previous sensory input (sensory dynamics, body dynamics, and physical relationships) retained by the central nervous system (CNS) (Reason & Graybiel, 1973). This structure has been the basis of observer models (Bos & Bles, 1998; Bos, Bles, & Dallinga, 2002; Bos, Bles, & Hosman, 2001a, 2001b; Glasauer, 1992, 1993; Glasauer & Merfeld, 1997; Matsangas, 2004; Merfeld, 1995; Merfeld, Young, Oman, & Shelhamer, 1993; Oman, 1982). The overall theoretical concept was based on earlier work by Holst (1954) and Held (1961) regarding the mechanisms of the central nervous system to distinguish between self- and induced motion.

According to this theory, motion sickness is triggered by a conflict between the prevailing sensory inputs and those expected on the basis of past experience (Reason & Graybiel, 1973). As Reason noted, the causal factor of motion sickness is the incongruity among normally synergistic channels of information (principally the eyes, the vestibular system, and the proprioceptive receptors in the joints, tendons, and muscles) (Reason, 1978). Money (1970) suggested that the extent of motion sickness incidence related to various modes of transportation is determined in part by the frequency and acceleration response of the vehicle to its environment, the susceptibility of the individual, and the amount of recent exposure of the passenger or crew to a similar motion environment. The effect of motion on motion sickness severity was assessed by researchers at Wesleyan University (Alexander, Cotzin, Hill, Ricciuti, & Wendt, 1945a, 1945b, 1945c). However, it was not until decades later that the Human Factors Research, Inc. (HFR) series of studies clearly identified the effect of motion envelope characteristics (acceleration amplitude, and frequency) on motion sickness severity. The latter experiments showed that motion sickness incidence (MSI): a) can be induced by whole-body motion at the frequency range between slightly below 0.1 Hz to slightly above 0.5 Hz, and b) that, at any given frequency within this frequency range, MSI was a monotonically increasing function of acceleration (McCauley et al., 1976; O'Hanlon & McCauley, 1974). The maximum severity of symptoms occurred at approximately 0.167 Hz (a 6 sec period) of simple sinusoidal vertical acceleration, the latter considered the principal provocative stimulus at sea (Griffin, 1990; McCauley et al., 1976; Morales, 1949; O'Hanlon & McCauley, 1974). Lawther and Griffin's (1987) analysis showed that the sensitivity to motion sickness caused by vertical oscillation spans over the frequency range 0.083 to 0.6 Hz, and reaches its maximum at approximately 0.2 Hz. Other studies showed that motions other than vertical can be nauseogenic as well (Joseph & Griffin, 2008). Golding et al. (2001) found that the motion sickness maximum for horizontal translational oscillation also was around 0.2 Hz. Howarth and Griffin (2003) concluded that motion sickness can be induced by pure roll oscillation, but it will usually be less than the sickness associated with pure translational oscillation or the sickness associated with combined translation and rotation. Donohew and Griffin (2004) identified the

development of mild motion sickness from lateral oscillatory acceleration in the frequency range between 0.0315 and 0.8 Hz.

In 1998, Bles and colleagues proposed the Subjective-Vertical conflict theory (Bles, Bos, de Graaf, Groen, & Wertheim, 1998). The authors restated the conflict theory in terms of a conflict between the perceived vertical and the subjective vertical as determined on the basis of previous experience. The initial theory, as well as an extension including horizontal accelerations (SVH), has been successfully used to predict motion sickness on ships (Bos & Bles, 2000; Bos et al., 2002; Dallinga, Pinkster, & Bos, 2002; Khalid, Turan, Bos, & Incecik, 2011; Verveniots & Turan, 2002). More specifically, the SVH conflict model has been valid in seven out of ten trials with three vessels (Khalid, Turan, Bos, Kurt, & Cleland, 2010).

In general, increase in acceleration magnitude of the nauseogenic frequency of oscillation and in the duration of exposure over several hours leads to increased sickness.

3. Effects on Performance

There is extensive research on the effects of motion sickness on cognitive performance (refer to the reviews by Baker, 1966; Colwell, 1989; Hettinger et al., 1990). The discussion of motion sickness has its origin with the ancient Greeks. What is now described as “motion sickness” has its roots in the original term nausea (“*ναυτία*”), which is still used in modern Greek. This term is derived from the word “*ναυς*” meaning “ship.” However, a more systematic approach to the study of motion sickness began during World War II.

Baker (1966) conducted a review of motion sickness research. He assessed a large volume of research and noted earlier reviews by McEachern and colleagues (1942), McNally and Stuart (1942), Schwab (1942), Birren (1949), Morales (1949), Tyler and Bard (1949), Wendt (1951), and Chinn and Smith (1955). In summary, Baker concluded that it was generally agreed that motion sickness impairs performance, although evidence suggested that motivation ameliorates this deterioration. He also noted that performance tests administered immediately after exposure to motion where emesis occurred either failed to distinguish or showed a trivial difference in performance. Overall, the author

concluded, “In view of the quantity of literature on motion sickness, it is with a feeling of growing incredulity that one gradually comes to realize that there is virtually no pertinent, documented information concerning the effects of either motion sickness or of motion upon human performance” (Baker, 1966, p. 2).

Following the efforts reported by Baker, research in the Pensacola Slow Rotation Room (SRR) exposed participants to rotational environments between 1.7 and 10 rounds per minute (rpm) constantly for a number of days (Clark & Graybiel, 1961; Graybiel et al., 1965; Guedry, Kennedy, Harris, & Graybiel, 1964). Subsequent to emesis, performance did not deteriorate in card sorting, dial setting, and arithmetic computation. The researchers attributed the observed variance in performance primarily to changes in motivation.

Later, in a study simulating sea motion conditions, experienced sailors performed a number of cognitive tasks (Abrams, Earl, Baker, & Buckner, 1971). The results did not reveal any performance decrements in tasks such as target classification, memory tests, sonar target detection, and turn count tests. It is interesting to observe, though, that these results were confounded by significant practice effects. The authors reported that performance continued to improve with repeated testing. Other studies reported a deleterious effect of motion sickness on the number of correct responses in arithmetic computation (Brand, Colquhoun, Gould, & Perry, 1967; Money, 1970).

Wiker and Pepper (1978) assessed performance on a small monohull vessel and found that performance results were not indicative of significant performance degradation while underway, compared to the in-port condition. However, the researchers made two interesting comments. First, they noted that their results were confounded by significant practice effects. Second, the performance deterioration they observed, although not at a significant level, could be the result of both biodynamic and physiological interference. These researchers also cited a study conducted by Sapov and Kuleshov (1975) on the effects of a six-week crew exposure to actual ship motion. Results showed significant decrements in mental and professional performance when steaming in open seas as compared to calm waters. Improvements in performance occurred later at sea, but performance generally remained below baseline. Professional efficiency was measured

by comparing the speed of performance on professional tasks with that of established “norms.” The researchers attributed the reduction in mental and professional efficiency to large increases in error rates (reduction in quality of work), not to a reduction in the rate of task completion (quantity of work). Unfortunately, these results do not distinguish whether the observed deterioration is associated with vessel motion, motion sickness, or both.

In another study regarding the effects of simulated Surface Effect Ship (SES) motions on crew habitability, the authors evaluated the participants’ capability to perform operational-like tasks under various types of motion conditions (Malone, 1981; O’Hanlon et al., 1977). The simulated motion characteristics approximated the motion of a 2000-ton Surface Effect Ship (SES), a predecessor program to the Landing Craft, Air Cushioned (LCAC). The researchers concluded that motion *per se* had no effect on cognitive performance. However, they noted (p. 22), “it seems that motion sickness is the limiting factor in determining how men can be expected to perform monitoring tasks (and probably most other tasks) during 24- and 48-hour exposures to similar motion environments.” As reported (O’Hanlon et al., 1977, p. 6),

Motion sickness was generally incapacitating to the subjects, rendering most incapable of performing all but the simplest experimental tasks. Moreover, sick subjects tended to exercise their prerogative of leaving the test environment, in some cases long before the scheduled completion of their motion exposure. [...] Most sick subjects could not, or would not, perform most experimental tasks and usually left the test environment after a variable period.

Wiker, Pepper, and McCauley (1980) conducted a study at sea, evaluating performance on three different vessels. Results showed significant decrements in performance in one of the vessels on five of the six tasks used. The observed task decrements between dockside and underway on a 95-foot patrol boat were associated with increases in motion sickness severity scores, changes in physiological indices of motion sickness, and acceleration characteristics related to motion sickness incidence. However, the researchers noted (p. 222), “whether performance was affected directly by the accelerations endured within the test compartment, or as a result of motion sickness provoked by the accelerations, cannot be objectively determined given the degree of

colinearity between [motion sickness scores] and cabin accelerations.” Nevertheless, they offered an explanation for why they thought that deterioration should be attributed to motion sickness. It is interesting to observe that in this study the quantity of performance was reduced, not the quality. This conclusion is the opposite of the Sapov and Kuleshov (1975) findings.

A 1985 study focused on identifying the impact of nineteen stressors on target detection performance of sonar operators at sea. Motion sickness was identified as 16th for submariners, 10th for surface operators, and 14th for helicopter operators (1st was the most adverse impact, 19th the least) (Wylie, Mackie, & Smith, 1985). Based on these subjective evaluations, the researchers conducted a literature review of the existing state of knowledge regarding these stressors and task performance. They concluded that the state of knowledge on motion and motion sickness’s effect on sonar operators’ performance was insufficient (Mackie, Wylie, & Smith, 1985).

In a review conducted in 1990, the authors evaluated more than 50 studies, some of which have been already reported herein (Hettinger et al., 1990). The authors concluded that the existing research did not provide conclusive results as to the effect of motion sickness on cognitive performance. Colwell (2000), by contrast, addressed performance at sea through self-reports. Almost half of the responses reported some degree of motion sickness, as well cognitive and physical problems in completing tasks in rough seas.

In his work to address performance in a moving environment, Wertheim concluded that motion *per se* does not affect cognitive performance (Wertheim, 1996, 1998). However, he noted that performance decrements are expected to occur when motion sickness develops. Such detrimental effects have been observed in studies related to short-term memory (Bos, 2011; Dahlman, Sjörs, Lindstör, Ledin, & Falkmer, 2009), in command and control tasks (Cowings, Toscano, DeRoshia, & Tauson, 2001), or in visual search (Golding & Kerguelen, 1992). A study with participants performing a memory and recollection task under mild motion sickness, however, failed to identify systematic performance effects (Bos, MacKinnon, & Patterson, 2005).

The literature addressed shows that the effect of motivation and task involvement in motion sickness severity has been noted in the past. Birren (1949) pointed out that most people who experience transient motion sickness can exert themselves sufficiently to perform adequately when necessary. Kennedy and colleagues proposed that the effect of mental activity on motion sickness should be explored (Kennedy, Moroney, Bale, Gregoire, & Smith, 1971). Griffin (1990) noted that motion sickness literature includes various reports of the beneficial effect of mental activity in minimizing sickness, but he did not cite any corresponding research. Correia and Guedry (1966) provided evidence that being involved in a mental task may alleviate motion sickness effects. Dobie and his colleagues showed that encouragement to suppress symptoms (“cognitive counseling”) increases tolerance (Dobie, May, Fischer, Elder, & Kubitz, 1987; Dobie, May, Fisher, & Bologna, 1989), whereas evidence (anecdotal data and subjective reports since WWII) suggest that even sick individuals can continue performing acceptably if highly motivated (Baker, 1966; Birren, 1949; Greenberg, 1946; Tyler & Bard, 1949).

In general, many researchers have postulated that the lack of performance decrements from motion sick individuals can be attributed to psychological reasons. However, clinical studies provided mixed results regarding the utility of placebo on nausea symptoms (Hróbjartsson & Gøtzsche, 2001; McCauley, Royal, Shaw, & Schmitt, 1979).

Finally, a recent study found that motion sickness severity can be reduced by performing a mental task (Bos, 2011). This finding provides evidence that cognitive workload interferes to some extent with motion sickness severity.

Unfortunately, our review failed to identify any studies on the effect of motion sickness *per se* on cognitive multitasking performance, with the exception of a recent study that evaluated cognitive performance during a nine-day period at sea (Valk, Munnoch, & Bos, 2008). Two cognitive tasks were used, a vigilance and tracking dual task, and a more complex multi-task. Results indicated that vigilance and tracking tasks are more sensitive to detrimental effects of ship motion and sea-sickness. The researchers postulated that this effect might be caused by either the motion itself or sea-sickness.

This review reveals a number of points that either provide insight or lead to concern: a) cognitive performance is not affected by motion *per se*, b) earlier research regarding motion sickness effects on cognitive performance provides valuable insight, but in many cases fails to provide conclusive results because of methodological issues, c) it is generally accepted that severe motion sickness will lead to performance decrements or cessation of performance, possibly due to reduced motivation, and, d) there are very few studies addressing the relationship between motion sickness and multitasking cognitive performance.

4. The Effect of Motion Sickness on Performance Improvement

Earlier motion sickness research viewed practice effects, that is, performance improvement over time, merely as a nuisance to the researcher (Abrams et al., 1971; Wiker & Pepper, 1978) because practice confounded or masked the effects of motion sickness on performance. We failed to identify any studies exploring the effects of motion sickness on practice effects, learning/skill acquisition (D. M. Johnson, 2005; Kolasinski, 1997, p.151), or reminiscence. Initially proposed by Ballard (1913), the latter technical term refers to performance improvement of a partially learned task in the absence of actual practice (Eysenck & Frith, 1977, p. 3). Reminiscence is associated with the “off-line,” i.e., after practice, consolidation of an acquired skill leading to performance improvements (Robertson, Pascual-Leone, & Miall, 2004).

Researchers have postulated that symptoms experienced in a simulator may compromise training through distraction and decreased motivation (McCauley, 1984). A later effort identified the beneficial effect of training under stress, however this study did not investigate the effect of motion sickness in training (McClernon, McCauley, O'Connor, & Warm, 2011).

5. Motion Sickness as a Stressor

Stress can be defined as a process by which certain environmental demands evoke an appraisal process in which perceived demand exceeds resources and results in undesirable physiological, psychological, behavioral, or social outcomes (Salas, Driskell, & Hughes, 1996). Stress induces significant cognitive effects in decision making and

attention and leads to distraction and increased reaction times (Bourne & Yaroush, 2003; Salas et al., 1996). Simple tasks needing automated responses will suffer less from stress than will responses in a complex task with underlying cognitive control (Yerkes & Dodson, 1908). Furthermore, stress effects may lead to a reduction of available attentional capacity (psychological adaptability) (Hancock & Warm, 1989).

Ideas regarding the relationship between motion sickness and cognitive resources are not new. Kennedy and colleagues (Kennedy, Berbaum et al., 1987, p. 11) noted that analogous ideas have existed for a long time, tracing back to Graybiel's (1968, 1969), "functional reserve," Teichner's (1958) "distraction principle," and Sherrington's (1906) "competition for the final common path." Focusing on adaptation and drowsiness associated with motion sickness symptoms, Kennedy and colleagues (1987, p. 11) suggested that "adaptation occurs in the form of new connections. These new connections occur at some cost—some penalty [...] This may help to explain why people get drowsy in connection with motion sickness; indeed, why they are drowsy following long-term car rides or train trips [...] Specifically, if the body undergoes extreme duress, and has gone into the 'I am poisoned' mode, it taps available resources." The later comment is based on an evolutionary hypothesis regarding motion sickness (Treisman, 1977). According to this theory, the perturbations in the spatial frameworks defined by the visual, vestibular, or proprioceptive inputs may be produced by motion, as well as by disturbances in sensory input or motor control produced by ingested toxins. Therefore, the function of emesis may be to rid the individual of ingested neurotoxins. Its occurrence in response to motion would be an accidental by-product of this system.

Later research provided some experimental validation of this postulation. Research has shown that body postural control is not an entirely automated process. Attentional resources that could otherwise be diverted to cognitive functions are allocated to functions such as controlling body sway and to accurately monitoring changes in bodily orientation (Andersson, Hagman, Talianzadeh, Svedberg, & Larsen, 2002; Yardley, Gardner, Lavie, & Gresty, 1999). The need for cognitive resources also has been identified in the processing and integration of vestibular and ocular motor sensory information (Talkowski, Redfern, Jennings, & Furman, 2005). The authors speculated

that interference with cognition occurs as a result of the sensory integration required for resolving input from multiple sensory systems. Disorientation and vertigo interfere with concurrent cognitive processes (Gresty & Golding, 2009; Gresty, Golding, & Nightingale, 2008).

Thus, far, we have addressed the effect of motion sickness on attentional resources, by diminishing the availability of these resources. Overall, these studies show that postural control, sensory integration, and disorientation require cognitive and attentional resources and cannot be considered automatic (Norman & Shallice, 1986; Schneider & Shiffrin, 1977). According to the neural mismatch theory, motion sickness is associated with an erroneous signal from the integration of information from sensory systems related to postural control; disorientation and ataxia are symptoms associated with motion sickness.

However, anecdotal evidence and research has also identified an opposing relationship. Being involved with a mental task may decrease the severity of motion sickness (Correia & Guedry, 1966, 1967; Graybiel, 1968; Guedry, 1964; Hill, 1936; Reason & Brand, 1975, p. 71). A recent study showed that motion sickness's severity can be reduced a) by introducing a high frequency vibration along with the nauseogenic, low-frequency motion, and b) when performing a mental task (Bos, 2011). The author concluded that additional head vibration can reduce motion sickness, in the same manner as mental distraction caused by a cognitive task.

Therefore, motion sickness seems to interfere with concurrent cognitive activity, and performing cognitive tasks while motion sick becomes more difficult because of the reduced availability of cognitive resources. In this case, "availability" may refer to: a) whether resources are freely available, or b) whether resources will be centrally de-allocated by a higher executive function from "contemplating on [motion] sickness" (Bos, 2011), and can thus be re-allocated to a task.

B. SOPITE SYNDROME

It has long been known that symptoms like drowsiness, apathy, lassitude, lethargy, and lack of interest in the task or the environment occur in nauseogenic motion

conditions (Byrne, 1912; Hill, 1936; Reason & Brand, 1975, p. 46). Studies conducted in a slow rotating room (SRR) reported depression and sleepiness (Graybiel, Clark, & Zariello, 1960). Reason and Brand (1975, p. 47) reported an unpublished study involving a three-day exposure to angular accelerations where drowsiness was persistent and overwhelming (Reason & Graybiel, 1971). These symptoms, though, often have been considered as minor or secondary when compared to others like nausea and vomiting (Schwab, 1954; Steele, 1961).

It was not until 1976, that Graybiel and Knepton (1976) changed the perspective by defining “sopite syndrome.” The term describes a symptom-complex centering on drowsiness and lethargy related to motion sickness. Sopite syndrome is associated with drowsiness, yawning, disinterest and disinclination to work, lack of participation in group activities, mood changes, sleep disturbances, and mild depression. Depending on the stimulus, sopite syndrome may be the only reported manifestation of motion sickness (Graybiel & Knepton, 1976; Kennedy, Lane, Berbaum, & Lilienthal, 1993; Mead & Lawson, 1997). Soporific symptoms have been shown to appear before nausea and to remain after the cessation of a nauseogenic motion stimulus (Dobie, 2003; Lawson & Mead, 1998). It has been suggested that sopite syndrome can lead to inefficiency and being accident prone, and that it could have profound effects in transport environments where, for other reasons, sleep disturbances exist (Graybiel & Knepton, 1976; Lawson & Mead, 1998).

Sopite syndrome is important operationally for a number of reasons: a) a person with sopite syndrome may have degraded performance, but may not be identified as motion sick, b) drugs for nausea may not improve degraded performance due to sopite effects, and c) medication effects might exacerbate existing drowsiness due to sopite syndrome (Buckey & Buckey Jr., 2006; Graybiel & Knepton, 1976; Lawson & Mead, 1998). Soporific effects are operationally important because they are common and frequent. Research has shown that drowsiness is among the most frequent symptoms associated with motion sickness (Cowings et al., 2001). Our review of the literature did not identify any studies related to the adaptation process for soporific symptoms.

C. MOTION SICKNESS DETECTION AND MEASUREMENT TOOLS

1. Self-reporting Tools

Subjective assessment of motion sickness has a long history. Self-reporting tools are used to evaluate two distinct issues, individuals' susceptibility to motion sickness, and the severity of motion sickness symptoms at a given time. For both issues, a number of survey tools have been proposed.

Given the current state of research, motion sickness susceptibility can be predicted to some degree through the motion history of the participants (Kennedy, Fowlkes, Berbaum, & Lilienthal, 1992). Individuals who often feel nausea symptoms in provocative real or apparent motion are more likely to develop symptoms in the future. However, self-rating of motion sickness comes at a cost. Although pre- and post-test questionnaires of motion sickness symptomatology have shown their usefulness in laboratory and applied research, they are shown to be biased by demand characteristics with post-session self-rated motion sickness severity being related to pre-session administration (Young, Adelstein, & Ellis, 2006, 2007).

An often used tool for the assessment of motion sickness severity is the Pensacola Motion Sickness Questionnaire—MSQ (Kellogg, Kennedy, & Graybiel, 1965; Kennedy, 1975), which is paired with the Motion History Questionnaire (MHQ). Further development of the MSQ focused its use with on simulators and led to the Simulator Sickness Questionnaire (SSQ) (Kennedy, Fowlkes, Berbaum, & Lilienthal, 1987; Kennedy et al., 1993; Kennedy, Lilienthal, Berbaum, Baltzley, & McCauley, 1989; Lane & Kennedy, 1988), which was developed to assess motion sickness in simulators and virtual environments. The SSQ contains a symptom rating list consisting of 26 symptoms which are self-reported by the participants (none, slight, moderate, and severe). These ratings contribute to three subscale scores (nausea, visuomotor discomfort, and disorientation). The ratings also are used for the weighted Total Severity (TS) score. The SSQ, as well as the MSQ, are administered as a pre- and post-test measure (Kennedy et al., 1993).

Subjective ratings of well-being are considered “the single most valuable source of information about the subject’s condition” to assess motion sickness severity and symptoms (Reason & Brand, 1975, p. 82). Based on the initial Reason and Brand Motion Sickness Susceptibility Questionnaire (MSSQ) (Reason, 1968; Reason & Brand, 1975), Golding proposed a revised version (Golding, 1998). The revisions resulted in improved scoring. The single MSSQ score ranges from 0 for no problems to 222 for severe problems. For a normal population, the 50th percentile is reached at approximately MSSQ 40. Compared to other motion sickness studies, the predictive validity of MSSQ for motion sickness tolerance using laboratory motion devices averaged $r=0.45$.

Motion Sickness Assessment Questionnaire (MSAQ) is another survey tool for the assessment of motion sickness severity (Gianaros, Muth, Mordkoff, Levine, & Stern, 2001). The MSAQ includes four subscales (Gastrointestinal, Central, Peripheral, and Sopite-related). The linear combination of the subscale scores leads to the overall motion sickness score. Overall scores from the MSAQ correlate strongly with the corresponding scores from the Pensacola Diagnostic Index (PDI) ($r=0.81$, $p < 0.001$) and the Nausea Profile (NP) ($r = 0.92$, $p < 0.001$). Scores from the subscales of the MSAQ and the NP were also correlated (Gianaros et al., 2001; Graybiel, Wood, Miller, & Cramer, 1968; Muth, Stern, Thayer, & Koch, 1996).

2. Psychophysiological Tools

The Autonomic Nervous System’s (ANS) psychophysiological responses have been extensively used for the detection of motion sickness. However, the psychophysiological variables are strongly idiosyncratic with no single physiological variable dominating in the prediction of motion sickness (Cowings, Naifeh, & Toscano, 1990; J. C. Miller, Sharkey, Graham, & McCauley, 1993). The following paragraphs will focus on the psychophysiological metrics to be used in this study. For a useful review of physiological correlates of motion sickness, see Harm (1990).

a. Electrocardiography (ECG or EKG)

Many studies have shown that cardiac activity changes with the development of fatigue (Hayashi, Minamitani, & Shin, 1997), stress (Hoover & Muth,

2004), and motion sickness symptom development. As motion sickness severity escalates, a gradual sympathetic activation leads to heart rate increases (Cowings, Suter, Toscano, Kamiya, & Naifeh, 1986; Holmes & Griffin, 2001; J. C. Miller et al., 1993; Oinuma, Hirayanagi, Yajima, Igarashi, & Arakawa, 2004; Stout, Toscano, & Cowings, 1995), and cardiac parasympathetic activity declines (Gianaros et al., 2003). High motion sickness susceptibility is associated with increased low-frequency (LF) Heart Rate Variability (HRV) power, decreased high frequency (HF) HRV power, and increased LF:HF ratio in these participants ($p < 0.05$) (Oinuma et al., 2004; Yokota, Aoki, Mizuta, Ito, & Isu, 2005).

b. Electrodermal Activity (EDA)

Differences in electrodermal activity (or skin conductance level, SCL) have been associated with motion sickness development (Cowings et al., 1990; J. C. Miller et al., 1993), and drowsiness (Dureman & Boden, 1972). One study noted that phasic skin-conductance responses recorded at the forehead site were the most sensitive physiological correlates of motion sickness induced by viewing an optokinetic rotating drum (Wan, Hu, & Wang, 2003). Nevertheless, other studies failed to identify significant associations between nauseogenic symptoms and EDA (Chung et al., 2007). Possible reasons for these non-findings may be the strong idiosyncratic attribute of these responses and that skin conductance has been associated with other responses, such as emotional arousal, anxiety, or stress (Kavanagh, 2005; Perala & Sterling, 2007).

c. Electrogastrography (EGG)

Electrogastrography (EGG) is an objective, noninvasive, measure of gastric myoelectrical activity. The use of this method is based on a shift in the dominant basal electrical activity, where there is an increase in the 4 to 9 cpm activity (tachygastric), and a decrease in normal 3 cpm (0.05 Hz) activity (although the latter might not be observed in some individuals (Tokumaru, Mizumoto, Takada, Tatsuno, & Ashida, 2003). The literature is inconclusive as to the diagnostic value of EGG in identifying motion sickness. A number of studies have shown that tachygastric is a useful

tool in motion sickness research (Andre, Muth, Stern, & Leibowitz, 1996; Holmes & Griffin, 2000; Hu et al., 1999; Hu & Stern, 1998, 1999; Imai, Kitakoji, & Sakita, 2006). However, other studies failed to support that claim or showed that other physiological measures may be more sensitive in detecting simulator sickness (J. C. Miller et al., 1993). For a review of EGG findings related to motion sickness, see Cheung and Vaitkus (1998) and Harm (1990).

The effect of food ingestion on EGG patterns in nauseogenic environments also is inconclusive. Although, one study did not find a significant effect of yogurt ingestion in EGG patterns when participants were subjected to stressful Coriolis stimulation (Stewart, Wood, & Wood, 1989), others showed an increase in the 3 cpm activity after the meal (Uijtdehaage & Stern, 2007), or after fluid intake (Hu, Lagormarsino, & Luo, 1998). The latter study found that tachygastria was significant in all experimental conditions but was not affected by fluid intake.

D. CLASSIFICATION OF MOTION EFFECTS

Motion sickness is a behavior that emerges as a physiological response to nauseogenic stimuli, i.e., real or apparent motion. Therefore, an investigation of motion sickness should consider the expected effects of motion, motion sickness, as well as other expected phenomena like practice effects and skill acquisition.

Wertheim (1998) classified the effects of environmental motion into two main categories, general and specific effects. Referring to any task or performance, general effects may be of a motivational nature (from motion sickness), of an energetic nature (motion-induced fatigue caused by the continuous muscular effort to maintain balance), or of a biomechanical nature (e.g., interference with task performance because of a loss of balance). Special effects, on the other hand, refer to interference with specific human abilities distinguished in three classes of tasks on the basis of their underlying skill components, a) cognitive tasks (e.g., attention, memory, pattern recognition), b) motor tasks (e.g., manual tracking, fast button pressing reactions), and c) perceptual tasks (e.g., visual or auditory detection).

Wertheim concluded that motion does not directly affect cognitive skills, whereas motor control is biomechanically affected to some extent, depending on the task and the type of motion. This classification is depicted in Figure 1.

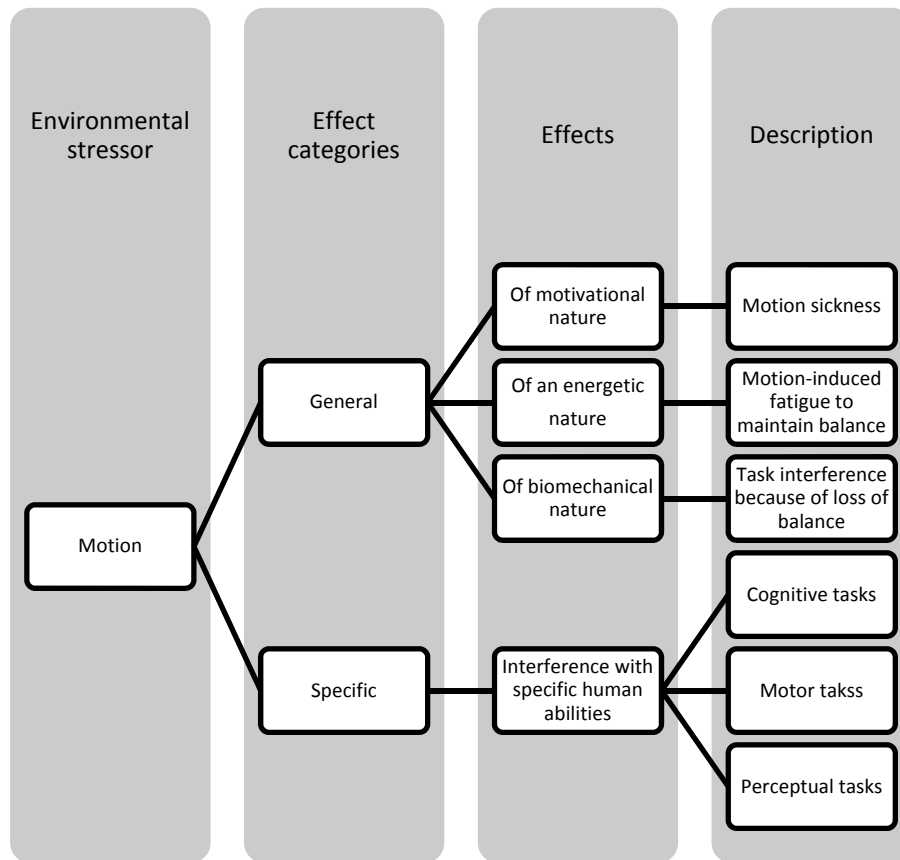


Figure 1. Classification of motion effects on performance (derived from Wertheim, 1998).

Another classification divides motion effects into direct and indirect (Colwell, 2005). Direct effects have an immediate impact on performance (biodynamic effects, loss of balance) while indirect effects impact performance through their symptoms (fatigue, motion sickness). Research has not identified any direct effects of motion on cognitive performance (Holcombe-Conwell & Holcombe, 1996; Wertheim, 1998).

E. SUMMARY

The literature review elucidates some interesting issues.

- Self-reporting tools are typically used to evaluate individuals' susceptibility to motion sickness and the severity of motion sickness symptoms at a given time.
- Although used in motion sickness research, the psychophysiological measures are strongly idiosyncratic. No single physiological variable dominates in the prediction of motion sickness signs or symptoms across individuals.
- Many studies fail to identify significant effects of motion sickness on performance due to three main reasons: a) non-stabilized performance (practice effects), b) lack of a control group, and c) use of a control group where participants perform in static conditions.
- There is a gap in the assessment of the effects of motion sickness on multitasking performance.
- There is a lack of adequate research regarding mild motion sickness and sopite syndrome. More specifically, a gap is identified in the experimental assessment and quantification of mild motion sickness/sopite syndrome effects on cognitive performance.

Given the aforementioned gaps in research, we will investigate the effect of mild motion sickness and sopite syndrome on multitasking cognitive performance. The following hypothesis guides this study:

Null hypothesis— H_0 : Multitasking cognitive performance is not affected by mild motion sickness and soporific effects.

Research hypothesis— H_1 : Multitasking cognitive performance is significantly reduced by mild motion sickness and soporific effects.

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III. METHODS

A. OVERVIEW

The experiment was conducted at the Naval Postgraduate School (NPS) in three data collection periods. This chapter provides the basic information regarding the experiments. More detailed information will be included in the following chapters. The first period was January through February 2010. The second period occurred a year later. The third period was May through June 2012.

Given that the main focus of the experiment was to assess the effects of mild motion sickness and sopite syndrome on multitasking performance, we collected four categories of data:

- SYNWIN (Elsmore, 1994) multitasking performance data
- Motion sickness and sopite syndrome severity data
 - Psychophysiological (EGG, ECG, skin conductance)
 - Subjective assessments (MSAQ and SSS)
- Sleep data (subjective, from sleep logs)
- Demographic data to assess background information, including susceptibility to motion sickness and prior exposure to nauseogenic environments

The overall approach in this experiment is depicted in Figure 2. Connections between blocks show probable associations suggested by existing research or associations to be assessed in this study.

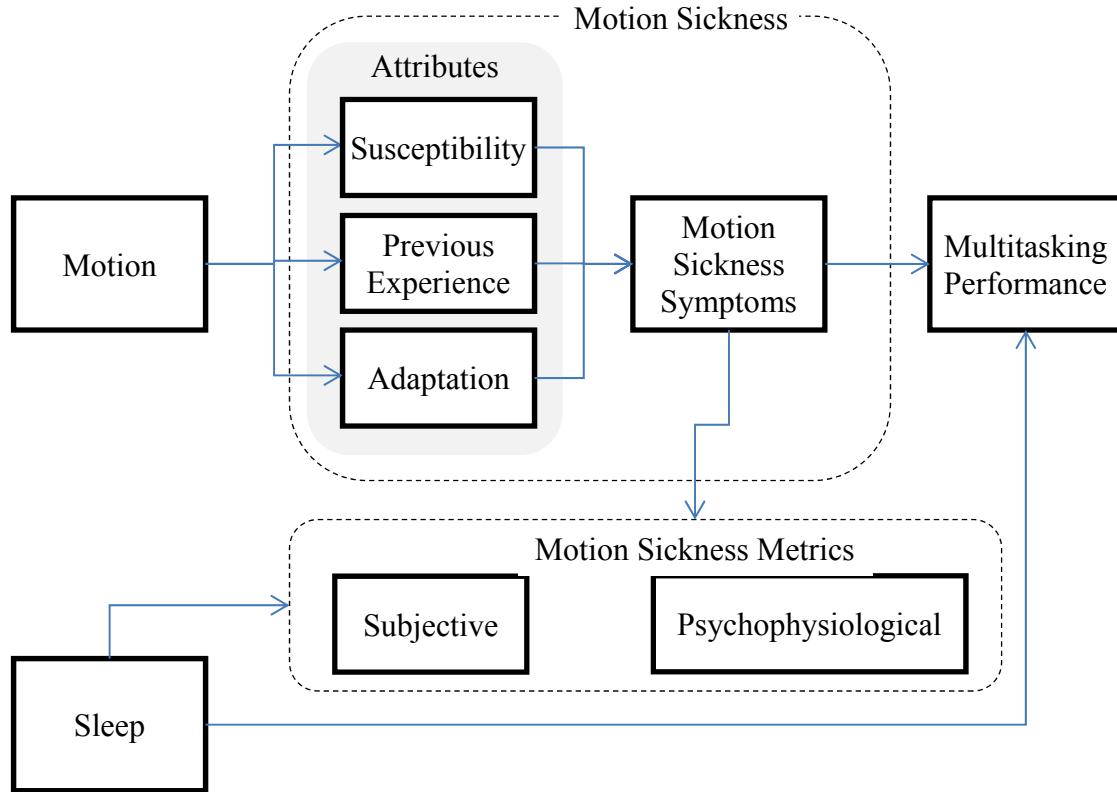


Figure 2. Experimental design overall approach.

The study protocol was approved by the NPS Institutional Review Board (IRB). Each participant provided written informed consent before participating in the experiment.

B. METHODOLOGY AND EXPERIMENTAL DESIGN

1. Participants

Individuals participating in the experiment were recruited by e-mail from the pool of NPS students, faculty and staff. All participants had normal vision and hearing and were screened before and during the study for illnesses or other issues that could affect their test performance.

a. Screening

(1) Sleep Disorders. To effectively address the relationship between sopite syndrome and multitasking performance, the effect of sleep must be considered. Individuals were screened for sleep disorders and excluded from the study since sleep disorders such as insomnia, sleep hypopnea or apnea, narcolepsy, or restless leg syndrome are known to interfere in sleep's restorative effects and induce excessive daytime sleepiness (EDS) (Pagel, 2009), which could be confused with sopite syndrome.

(2) Motion Sickness. Individuals with a history of gastrointestinal, cardiovascular, or vestibular disorders (labyrinthine defects, labyrinthectomies, etc.) were excluded from the study.

(3) Substance Intake. Research has shown that certain substances should be avoided during the day before the test session, such as melatonin (Graw et al., 2001) and alcohol (Greece et al., 2005). This information was provided to the participants in the beginning of the study; immediately before each experimental session, participants were asked whether they had used any of the substances.

(4) Other Health-Related Issues. In simulator experiments there are certain health issues that might affect participation, such as migraines (Viire & Bush, 2002) or photic seizures (Kasteleijn-Nolst Trenite et al., 1999).

b. Controlled Variables

We identified a number of variables that could affect multitasking cognitive performance or motion sickness severity. Our approach was to keep these variables constant across the three data collection sessions.

(1) Food and Fluid Intake. Participants were instructed about their food ingestion in the period preceding the data collection period (dinner and breakfast). Research is inconclusive regarding the effect of food ingestion on motion sickness symptoms. Food ingestion may lead to increased symptoms (Stewart et al., 1989), decreased symptoms (Levine, Muth, Williamson, & Stern, 2004), or no effect (Hu et al., 1998)). However, participants were asked to have a moderate dinner and breakfast,

without an excessive fluid intake during or after breakfast. Participants also were asked about consumption of caffeinated beverages.

(2) Time. The known effects of circadian rhythmicity on performance (Carrier & Monk, 2000) were controlled by scheduling each participant's two data collection sessions at the same time of day.

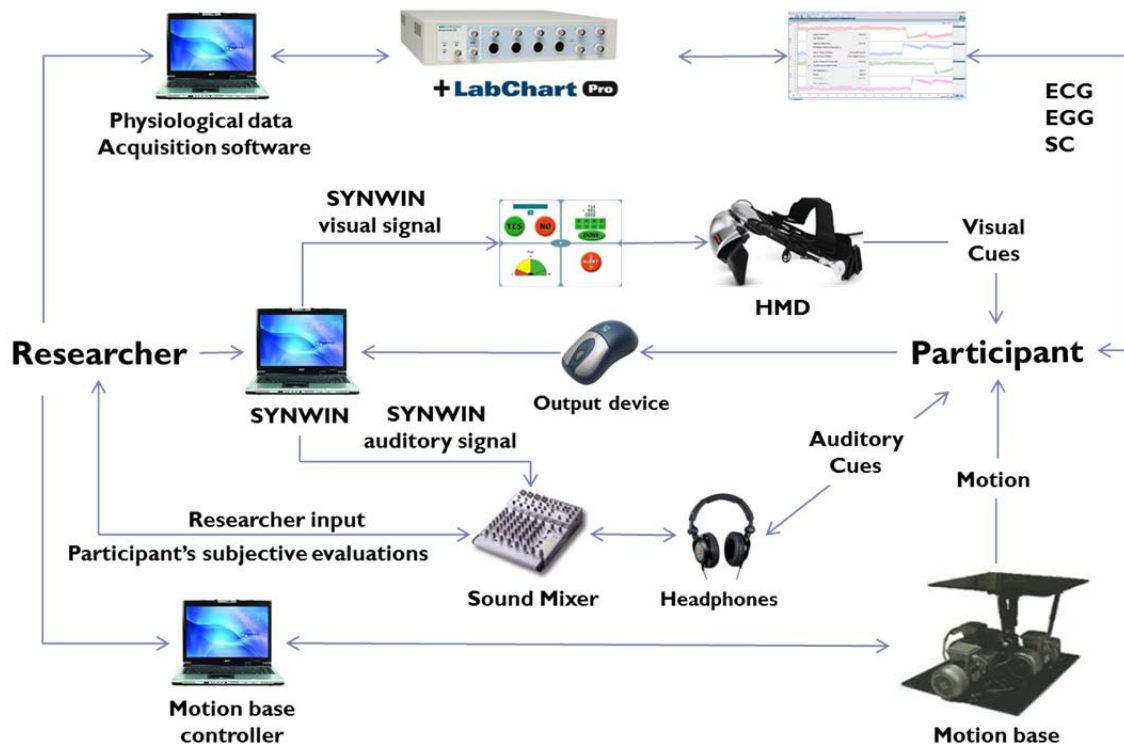
(3) Participant Position. Participants were seated and instructed to keep their head on the back of the chair. There are two underlying reasons for this instruction. First, it is known that exposure to real motion disrupts balance, increases postural sway, and in some cases, results in ataxia. This postural disequilibrium may pose a safety risk in standing participants. Special care was taken with the participants after the exposure to the motion stimulus in case ataxic after-effects were observed (Kennedy, Berbaum, & Lilienthal, 1997). On the other hand, head motion in a real motion environment may elicit Coriolis or pseudo-Coriolis effects (i.e., when the head is tilted during illusory self-rotation induced by moving visual stimuli) (Dichgans & Brandt, 1973; W. H. Johnson & Sunahara, 1996). Apart from the fact the free head movement will result in a variable that cannot be controlled, it is known that Coriolis effects result in rapid and drastic increase of motion sickness severity, a result which was not desired in this study.

2. Equipment and Instruments

a. Overview

The cognitive multitasking battery used in this study was SYNWIN (Elsmore, 1994) running on a Microsoft Windows laptop. During data collection, participants donned the head-mounted display system (HMD) eMagin Z800 3DVisor where the multitasking battery was projected. Instead of a laptop or a flat panel display, we chose the HMD solution to exclude any visual stimuli from the external environment.

None of the participants had prior experience in using the software or the HMD. Participants wore headphones that presented the SYNWIN tone stimuli. Responses were made with a mouse using the participants' dominant hand. Figure 3 illustrates the basic equipment layout.



b. SYNWIN

The multitasking battery used in the experiment was SYNWIN (™ Activity Research, Inc.), a computerized, multitasking neuro-psychological assessment software program. SYNWIN is the Windows version of SynWork1, a DOS-based program (Elsmore, 1994) simulating a work environment. Shown in Figure 4, the synthetic work environment is optimized for display screen resolution of 800 by 600 pixels, and responses are made by a mouse driving a cursor. SYNWIN does not emulate any specific work application, but it does contain generic elements of work-based activities that are common to a number of watch-standing jobs (Elsmore, 1994; Proctor & Wang, 1998). It includes up to four component tasks presented simultaneously: a memory search task, an arithmetic problem task (the only self-paced task in the battery), and two visual and auditory reaction tasks (see Figure 4).

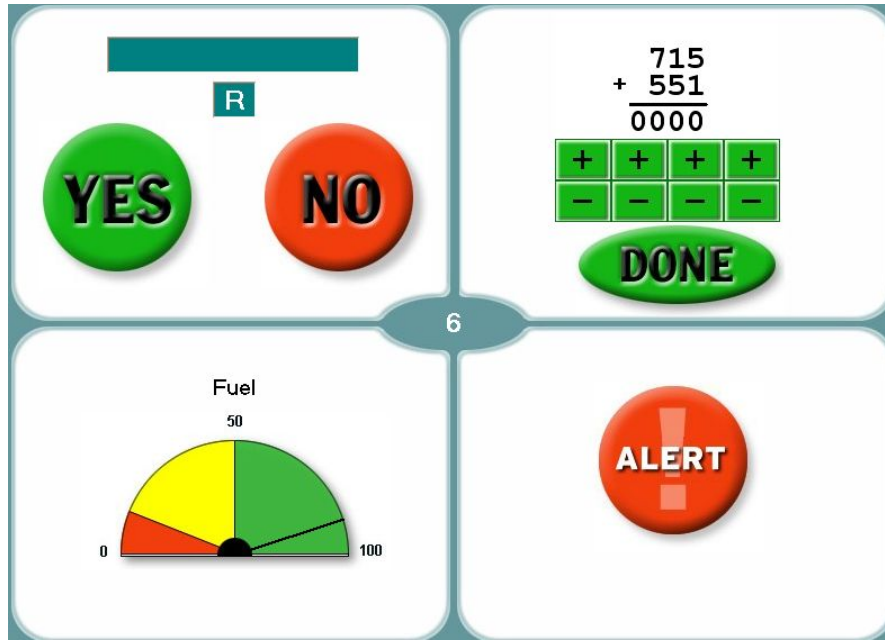


Figure 4. Screenshot of SYNWIN test.

The screen is divided into quadrants, each one allocated to a task. The small oval at the center of the screen contains the composite score for the ongoing session. The memory search task is presented in the upper left window, the arithmetic task in the upper right, the visual task is presented in the bottom left window and the auditory task in the bottom right. At the beginning of a memory task session, a list of letters is shown for five seconds in the upper box near the top of that window. After the letters disappear, a target letter is displayed in the middle box for 20 seconds. The participant responds by clicking on the *Yes* or *No* button to indicate whether the displayed letter was a member of the initial list of letters. Points are gained for a correct response whereas a point penalty is assessed for incorrect responses or whenever the participant chooses to review the initial list of letters again. In the self-paced arithmetic task displayed in the upper right window, participants sum two three-digit numbers by adjusting the value 0000 below the numbers. The adjustment is made by clicking on the plus and minus signs corresponding to each digit column. When ready, the participant clicks the *Done* button and new numbers appear. Points are added or subtracted for the correct or incorrect sum, correspondingly.

In the visual monitoring task (lower left quadrant) a pointer moves at a fixed rate from right (100 position) to left (0 position) across a background that resembles a fuel gauge. Participants must prevent the pointer from reaching 0 by clicking on the gauge to reset the pointer to the 100 position. More points are received for the pointer being as close to 0 as possible and points are lost for every second the pointer stays at 0. In the auditory monitoring task (lower right quadrant) participants must respond every five seconds to a higher-pitch target tone (regarded as positive) and ignore a lower-pitch tone (regarded as negative). Points are awarded (correct detection) when detecting the positive sound by clicking the *Alert* button before the next sound occurs. Points are subtracted on erroneously detecting the positive (*Miss*) or negative sounds (false alarm).

Each SYNWIN session was set to ten minutes duration. This “session” is referred to as a “ten-minute block” in the rest of the text, and should not be confused with the Experimental Session (ES), which includes six ten-minute blocks. All four SYNWIN tasks were conducted simultaneously. The number of letters in the memory task was set to five. In the auditory task, the frequency of the positive tone was 1025 Hz, whereas the negative tone was 1000 Hz. The probability of a positive tone was $p=0.200$. The rest of SYNWIN functional parameters were at their default values. The points added or subtracted per task were set according to the values in Table 1.

Table 1. Points added or subtracted per SYNWIN task.

Task	SYNWIN parameter	Set value
Memory (MT)	Correct YES	+10
	Correct NO	+10
	Incorrect YES	-10
	Incorrect NO	-10
	Failure to respond before next trial	-10
	Letter list retrieval	-10
Arithmetic (ArT)	Correct responses	+10
	Incorrect responses	-10
Visual (VT)	Reset (actual value determined by pointer position at reset)	Maximum = +10
	Pointer at the end of scale	-10
Auditory (AuT)	Correct detection	10
	False alarm	-10
	Miss	-10
	Quiet	0

As noted by Hambrick and colleagues (2010), there are no empirical demonstrations of the validity of SYNWIN for job performance. However, the four generic SYNWIN tasks address known cognitive resources and constitute a basis for the cognitive tasks commonly found in various work environments and occupations (Alluisi, 1967; Proctor & Wang, 1998). Watch standing on a ship, piloting an aircraft, or driving a wheeled vehicle all involve these generic tasks: monitoring visual displays for information; monitoring, reacting to, or prioritizing auditory warning signals; monitoring external visual information and reacting in cases of emergency; or using working memory for maintaining information. Based on earlier research (North & Gopher, 1976; Trankell, 1959), Hambrick and colleagues (2010, p. 1151) concluded that “there is reason to think that SYNWIN may prove useful as a predictor of performance in occupations such as pilot, where demands on multitasking are presumably high.”

It is interesting to assess the four tasks illustrated in Figure 3 from the perspective of the multiple resource theory (Wickens, 2002). The memory and the arithmetic tasks are associated with working memory capacity (Ashcraft, 1995; LeFevre, De Stefano, Coleman, & Shanadan, 2005; Turner & Engle, 1989). Being primarily perceptual, the visual and auditory monitoring tasks occur in different sensory modalities and assess distinct attentional resources (Wickens, 1980, 2002). Based on the working memory system model (Baddeley, 2003, 2004; Baddeley & Hitch, 1974), it was suggested that SYNWIN may engage the phonological loop (for maintaining the set of letters in the memory task and solving the arithmetic task), and possibly the visuo-spatial sketchpad for visualizing the position of the needle in the visual monitoring task while looking away from the gauge (Hambrick et al., 2010). Lastly, the manual component of SYNWIN (controlling the mouse) does not interfere with the four tasks (Wickens, 2002).

In each session, the objective was to obtain as many points as possible, while in the background SYNWIN records many performance measures (45 in total).

c. Physiological Data Acquisition System

The ADInstruments PowerLab 16/35 PL 3516 data acquisition system with the skin conductance response amplifier ML116 GSR and the Dual Bio amplifiers was used for physiological data acquisition. Collection and off-line analysis of physiological data was conducted by LabChart Pro 7.2 software.

d. Head Mounted Display

The eMagin Z800 3DVisor Head Mounted Display was used for the head mounted display. The apparatus provides approximately 40 degrees diagonal field of view, in two displays with 4:3 aspect ratio. The resolution was 800 by 600 pixels per display (SVGA).

e. Motion Base

The ASE Model 500–3 motion seat manufactured by Aeronautical Systems Engineering, Odessa, Florida, was used as the motion base. The apparatus uses 220-volt power to drive three separate motors. It is capable of the following motion characteristics.

Table 2. Motion base characteristics.

Degree of freedom	Limits of motion	Velocity (degrees per second)	Maximum Acceleration
Roll	+/- 15 degrees	+/- 50	300 degrees per second ²
Pitch	+/- 15 degrees	+/- 50	300 degrees per second ²
Vertical (heave)	+/- 2 inches	+/- 11	+/- 0.4 g

To ensure safety, there were two STOP buttons, either of which would immediately stop the motion stimulus. One button was located on the chair where the participant was seated and the other was within reach of the observing researcher.

The nauseogenic motion stimulus included the superposition of three independent 0.167 Hz sinusoidal motions. In the z-axis, the motion was set to +/- 2 inches displacement (heave). In the y and x axes, the motion was set to +/- 15 degrees roll and pitch, correspondingly.

f. Motion Sickness Assessment

Participants' susceptibility to motion sickness was assessed by the revised version of Motion Sickness Susceptibility Questionnaire—MSSQ (Golding, 1998). The severity of motion sickness and sopite syndrome symptoms was assessed by subjective and objective metrics. The primary subjective tool was the Motion Sickness Assessment Questionnaire (MSAQ) (Gianaros et al., 2001). We further evaluated soporific severity by the Stanford Sleepiness Scale—SSS (Hoddes, Dement, & Zarcone, 1972), asking participants to report their subjective assessment of their alertness after each ten-minute block.

The objective measurements included three psychophysiological metrics: gastric activity by EGG; EDA by skin conductance; and cardiac activity by ECG. Collection and off-line analysis of all physiological data was conducted by LabChart Pro 7.2 software.

(1) Electrogastrography (EGG). Gastric activity was assessed according to existing EGG procedures (Parkman, Hasler, Barnett, & Eaker, 2003). Data were obtained from three electrodes. The active electrodes were positioned below the left costal margin, between the xyphoid process and umbilicus. The reference electrode was positioned approximately 10 cm to the right of the abdominal midline and 5 cm above the umbilicus (Parkman et al., 2003).

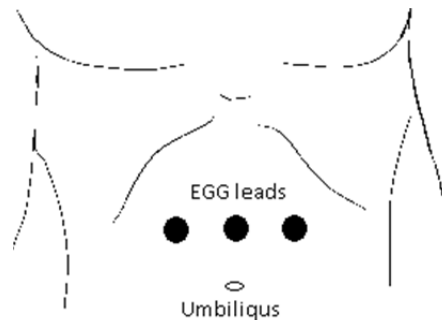


Figure 5. Placement of electrogastrography electrodes.

Cables were attached to disposable self-adhesive patches with embedded conducting gel. ECG cardiogram signals were sampled at 1000 Hz. Spectral analyses using fast Fourier transforms (FFTs) were conducted for each ten-minute block of EGG data. To remove baseline wandering or other high frequency noise, we implemented a band pass filter with low cut-off frequency of 0.02 Hz (1.2 cpm) and a high cut-off of 0.30 Hz (18 cpm). A Hann (cosine-bell) window was used to taper the EGG signal. After windowing, spectral density estimates were derived from FFTs within the frequency range of 1.2–18 cpm. The average power and the percentage of total power were then calculated for the tachygastic bandwidth (>4.0 cpm). The equations for the percentage of total power were calculated using the following basic equation:

$\text{Power\% in the frequency band} = (\text{band power} / 1.2\text{--}11 \text{ cpm power}) \times 100.$

Although not standardized, the tachygastric motility band was chosen based on existing literature (Chang, 2005; Parkman et al., 2003). A special note is needed regarding the frequency band between 9 and 11 Hz. According to Parkman et al. (2003), frequencies beyond 9 Hz are often interpreted as a respiratory artifact. Nevertheless, our initial analysis demonstrated that this EGG HF power is evident only in motion sick participants. Given that other researchers include this frequency band in their analysis, we decided to include the 9 to 11 Hz region in our analysis (Chang, 2005; Gianaros et al., 2001).

(2) Electro-Dermal Activity (EDA). Skin conductance (SC) is the reciprocal of skin resistance. SC was recorded by two electrodes placed on the volar surface of the distal phalanges of the index and middle fingers of the non-dominant hand. We calculated the average (SC M) of skin conductance in micro-Siemens (μS), per ten-minute block.

(3) Electrocardiography. An electrocardiogram (ECG) was obtained from three electrodes that were positioned according to Einthoven's Triangle configuration (Schamroth, 1990), as depicted in Figure 6.

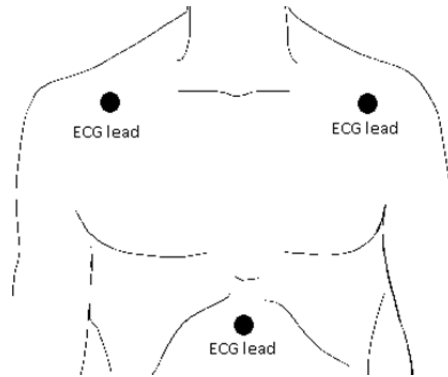


Figure 6. Placement of electrocardiography electrodes.

Cables were attached to disposable self-adhesive patches with embedded conducting gel. ECG cardiogram signals were sampled at 1000 Hz. Raw data were filtered to limit artifact occurrence and reduce baseline wandering and power line noise. We implemented a band pass filter with low cut-off frequency of 0.5 Hz and a high cut-off of 35 Hz. Artifacts were removed by setting the ECG detection algorithm with the following settings: a) Typical QRS width=80ms, and b) R waves are at least 300 ms apart.

After the detection of each QRS complex, normal-to-normal (NN) intervals were derived—intervals between adjacent QRS complexes resulting from sinus node depolarization (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

g. Personality

Personality assessment was conducted by the Neuroticism-Extroversion-Openness Five-Factor Inventory (NEO-FFI) (Costa & McCrae, 1992). Derived from NEO-PI-R, the NEO-FFI consists of 60 items used to score the five personality domains: neuroticism, extraversion, openness, agreeableness, and conscientiousness.

h. Sleep History

Sleep history was assessed by two validated survey instruments, the Epworth Sleepiness Scale—EPSS (Johns, 1991), and the Pittsburgh Sleep Quality Index—PSQI (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989). The EPSS is an eight-item scale used to diagnose sleep disorders. Research has shown that EPSS scores equal to or greater than 10 indicate a probable sleep disorder, whereas a score equal to or greater than 15 indicates excessive daytime sleepiness. The second instrument, PSQI, differentiates “poor” from “good” sleepers by measuring seven areas related to sleep in the previous month: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. Scoring is based on a 0 to 3 scale in which 3 reflects the negative extreme on the Likert Scale. A global sum equal to or greater than 5 indicates a poor sleeper whereas a score less than 5 indicates a good sleeper.

The Stanford Sleepiness Scale (SSS) (Hoddes et al., 1972) was also used in this study to assess alertness before the beginning of each experimental session, and after each ten-minute block. The participant assess his sleepiness by choosing one from eight states, ranging from “feeling active, vital, alert, or wide awake” to “asleep.”

The last instrument, the Morningness-Eveningness Scale (Horne & Östberg, 1976), was used to assess participants’ chronotype, an attribute of human beings related to whether they have a preference for waking earlier or later in the day. The scale includes 19 multiple-choice questions. Scores range from 16 to 86, with scores less than 42 corresponding to evening chronotypes and scores higher than 58 indicating morning chronotypes.

i. Study Questionnaires

Participants were screened for illness and health issues related to the experiment (Screening Questionnaire). Then, they filled out the Study Questionnaire including demographic questions (age, gender, weight, height, and motion experience), questions addressing the use of caffeinated beverages and medications, smoking habits, the EPSS (Johns, 1991), the PSQI (Buysse et al., 1989), the Morningness-Eveningness Scale (Horne & Östberg, 1976), Neuroticism-Extroversion-Openness (NEO) Five Factor Inventory (Costa & McCrae, 1992; McCrae & John, 1992), and the revised motion sickness susceptibility questionnaire (MSSQ) (Golding, 1998).

A question regarding smoking habits was included in the Study Questionnaire because research has demonstrated that smoking interferes with performance in a complicated manner (Tong, Leigh, Campbell, & Smith, 1977), either by enhancing cognitive performance (Foulds et al., 1996), or deteriorating performance when smokers abstain from nicotine (Rissling, Dawson, Schell, & Nuechterlein, 2007).

The physiological status of female participants has been included in the Study Questionnaire (i.e., pregnancy, menstrual cycle), because research has shown that the menstrual cycle affects female susceptibility to motion sickness (Matchock, Levine, Gianaros, & Stern, 2008). Given the hypotheses to be tested under this work, though, we decided to treat female and male data in the same manner.

The physiological state of the participants was assessed before each experimental session by a survey instrument (the Physiological Status Questionnaire) which included questions about health issues, EPSS, and the SSS. Alcohol was forbidden during the day before the experimental sessions, and was to be avoided during the remainder of the study. After each ten-minute SYNWIN block, participants provided a subjective evaluation of their physiological state by responding to the MSAQ and the SSS. The latter test was used to assess reduced alertness due to drowsiness, a symptom associated with sopite syndrome. Participants also rated the extent to which motion biodynamically interfered with their performance of the SYNWIN tasks.

3. Procedures

Participants were tested individually in a quiet room and were asked to concentrate on the tasks and perform their best on each test. Participants performed the SYNWIN battery while seated wearing the HMD where the computer-based neuropsychological battery (SYNWIN) was projected (see Figure 7).



Figure 7. Research layout.

Each individual participated in two one-hour data collection periods (each one called an “experimental session”) with an inter-session interval of seven days. Each experimental session consisted of six ten-minute SYNWIN blocks for a total of 60 minutes. None of the participants had prior experience with SYNWIN. They received approximately two to three minutes of initial practice to learn the basics of the tasks (the displays, the controls and the procedures associated with using SYNWIN). This initial learning session was completed only when the participant understood the instructions and felt accustomed to the SYNWIN layout.

We used a counterbalanced design with participants from 2010 and 2011 data collection sessions randomly assigned to one of two groups: “M-NM” for the sequence “motion – no motion,” and “NM-M” for the sequence “no motion – motion.” Participants were seated on a small motion platform and the motion stimulus was presented during the last four SYNWIN sessions (for example, group “M-NM” participants experienced the

motion stimulus only during SYNWIN sessions 3, 4, 5, and 6 of the first experimental one-hour session, where group “NM-M” participants experienced the motion stimulus only during SYNWIN sessions 3, 4, 5, and 6 of the second experimental one-hour session). All participants from the 2012 data collection were assigned to group “NM-NM” and did not experience motion in either session. For simplicity, in the text we refer to the first experimental session (ES) of group M-NM as “M-NM-1,” the second ES of group M-NM as “M-NM-2,” etc. Figure 8 depicts the experimental design.

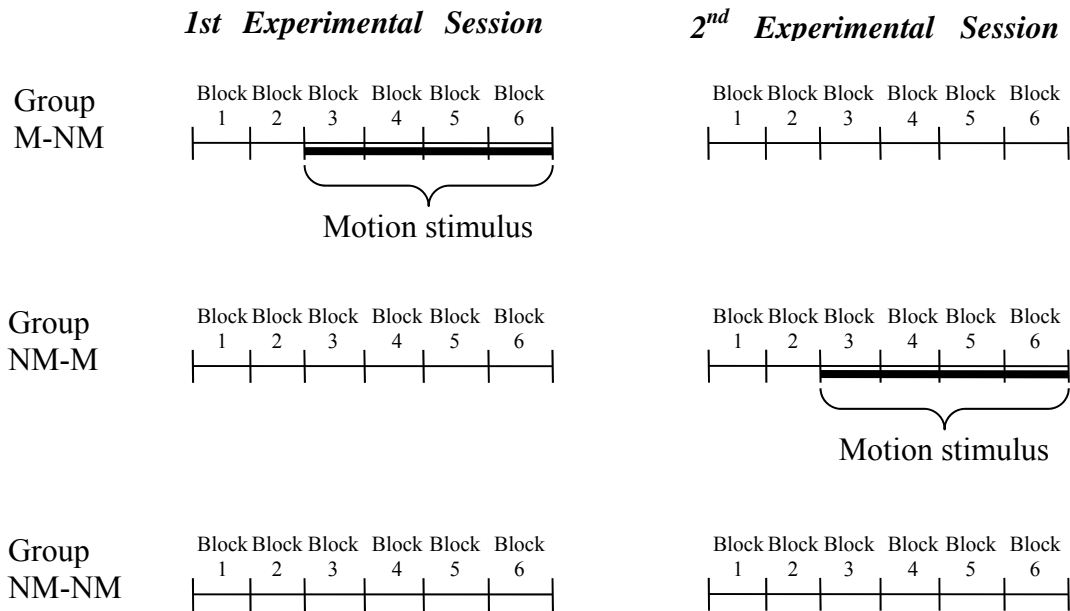


Figure 8. Experimental design.

Sleep data collection started approximately three days before the first experimental session and continued until the end of the last session (a total of ten days). Sleep evaluation was based on sleep logs kept by the participants.

During the sessions, the experimenter was located in the same room as the participant and environmental conditions were controlled (ambient temperature was kept constant between 68 and 69 degrees Fahrenheit; lights were out). To isolate possible environmental noise and to let the participants focus on the SYNWIN test, they wore headphones that presented the tone stimuli. The SYNWIN multitasking battery was set to run all four tasks simultaneously. Both experimental sessions were conducted at the same

time of day for each participant to control for circadian rhythmicity. Participants did not receive visual input from the external environment because they wore the HMD and the room was dark. The participants started the subsequent ten-minute SYNWIN block when they were ready to continue. This time ranged from one to three minutes.

4. A Linear Model of the Experiment

Based on existing classifications addressing the effects of motion on performance (Colwell, 2005; Wertheim, 1998), we developed a model to investigate the main focus of this study, i.e., the effects of motion sickness.

a. Initial Counter-balanced Experimental Design

Initially, the experimental approach was a crossover design (counterbalanced) incorporating 2 x 2 data collection. Participant group “M-NM” received motion in the first session, whereas participant group “NM-M” received motion in their second session. Such experimental designs (repeated measures design where different individuals receive treatments in different sequence) are affected by two potentially confounding factors, the carryover effect and the order or sequence effect (Neter, Kutner, Nachtsheim, & Wasserman, 1996; Quinn & Keough, 2002). The carryover effect occurs when a previous treatment influences behavior in a following treatment in the sequence (Bordens & Abbott, 2006b). The sequence effect refers to when measurements early in a sequence may be different from those taken later, irrespective of treatment (Neter et al., 1996). The sequence effect (the fact that different participants receive the treatments in a different sequence) was addressed by random assignment of participants to groups and by normalizing performance on a per participant basis.

The direct effects of motion are modeled by the term b_{ij} , representing the biodynamic effect on performance. We consider two components as direct effects, motion induced interruptions (MIIs) on balance, and the biodynamic interference on fine motor skills (for example, using the mouse). Regarding the MIIs, the participant was comfortably seated and strapped in the “bucket” type chair on the motion base, with arms at rest, so that balance issues are not a problem. On the other hand, research has shown

that motion may interfere with fine motor skills, but not always (Wertheim, 1998). Ballistic tasks or manual tasks with supported arms are effected minimally or not at all (McLeod & Poulton, 1980). In the experiment, all efforts were made to minimize the effect of motion on fine motor skills.

In this study physiological fatigue and motion sickness are considered indirect effects. Research has shown increased levels of physiological fatigue after using a mouse in static conditions for three hours (P. Johnson, Lehman, & Rempel, 1996). However, the literature review failed to identify corresponding research for shorter durations of mouse use, or research associating fatigue because of mouse use and performance deterioration. Physiological fatigue, as well as motion induced fatigue (MIF), was not expected to be an issue in the experiment because a) the participants were seated, with their arms at rest, using a mouse for only one hour, and b) participants stopped performing the task and using the mouse for a couple of minutes after the completion of each ten-minute block.

The effect of motion sickness on performance was the main focus of our experiment, and is modeled by the term m_{ij} . For a healthy individual, $m_{\text{No Motion},j} = 0$. The following model addresses the effects of motion and motion sickness on performance by differentiating between the main biodynamic motion effect (hence motion effect), and the main motion sickness effect. All two-way interaction terms were included.

$$Y_{ijh} = \mu + b_{ij} + r_{ij} + m_{ij} + (br)_{ij} + (bm)_{ij} + (rm)_{ij} + \epsilon_{ijh} \quad (1)$$

where:

i = treatment, where $i = \{\text{Motion, Static}\}$

j = experimental session, where $j = \{1,2\}$

h = participant

Y_{ijh} = performance of participant h in experimental session j under treatment i

μ = overall mean performance

b_{ij} = biodynamic effect on treatment i in session j

r_{ij} = effect of experimental session j under treatment i addressing the between-sessions practice effect

m_{ij} = effect of motion sickness in treatment i in experimental session j

$(br)_{ij}$, $(bm)_{ij}$, $(rm)_{ij}$ = interaction terms

ε_{ijh} = error term, where $\varepsilon_{ijh} \sim N(0, \sigma^2)$ [model assumption]

The following paragraphs provide more information regarding the interaction terms modeled.

- $(br)_{ij}$ is the interaction of treatment (motion, static) and experimental session. This interaction may take the following forms:
 - Over time, the individual develops a strategy to ameliorate the impact of motion on biomechanical aspects of the task. Therefore, during the second session, this skill may be better developed. On the other hand, a strategy under motion may interfere with performance under static conditions, and vice versa.
 - Motion during the first experimental session may have a beneficial effect on performance during the second session. The impact of such a motion stressor has not been addressed in the literature *per se*, although McClernon (2011) focused on this issue with a different stressor.
- $(bm)_{ij}$ is the interaction of motion sickness with the biodynamic effects of motion. Motion sickness is known to be associated with postural stability issues and ataxia (Benson, 2002; Takahashi, Takei, Saito, Okada, & Kanzaki, 1992; Villard, Flanagan, Albanese, & Stoffregen, 2008). Given that our participants were seated with their arms supported, this postural stability effect is assumed to be minimal.
- $(rm)_{ij}$ is the interaction of motion sickness and experimental session. This term describes the differential effect of motion sickness on performance between sessions.

Overall, the interactions between motion sickness, motion, and experimental session suggest that the use of a counterbalanced design is problematic. For this reason, it was decided to analyze the data independently for each motion session, and to add a control condition (NM-NM).

b. Implemented Analytical Approach

For the identification of motion sickness effect, the analytical approach we implemented was a between subject design under motion. Participants in the second session have already performed SYNWIN for one hour in their first (static) session, whereas participants performing SYNWIN in their first motion session have no prior knowledge of SYNWIN. Therefore, analysis independent for each session excludes comparisons between participants with different levels of proficiency in the multitasking battery. This approach simplifies equation (1) as follows.

$$Y_{\text{Motion},h} = \mu_{\text{Motion}} + b_{\text{Motion}} + m_{\text{Motion}} + (bm)_{\text{Motion}} + \varepsilon_{\text{Motion},h} \quad (2)$$

where:

$Y_{\text{Motion},h}$ = performance of participant h under motion

μ_{Motion} = mean performance in experimental session with motion

b_{Motion} = biodynamic effect of motion

m_{Motion} = effect of motion sickness under motion

$(bm)_{\text{Motion}}$ = is the interaction of motion sickness with the biodynamic effect of motion.

For the reasons already explained, $(bm)_{\text{Motion}}$ is assumed to be minimal.

$\varepsilon_{\text{Motion},h}$ = error term under motion, where $\varepsilon_{\text{Motion},h} \sim N(0, \sigma^2)$ [model assumption]

Based on $(bm)_{\text{Motion}} \sim 0$, equation (2) is simplified to the following form:

$$Y_{\text{Motion},h} = \mu + b_{\text{Motion}} + m_{\text{Motion}} + \varepsilon_{\text{Motion},h} \quad (3)$$

The analysis to address the effect of motion sickness on performance is based on two main methods: correlation analysis of performance scores versus MSAQ Total, and

performance comparison between Asymptomatic and Symptomatic participants. Modeling of the classification method leads to the following alternatives.

$$Y_{\text{Motion,Asymptomatic},h} = \mu + b_{\text{Motion}} + \varepsilon_{\text{Motion},h} \quad (3.1)$$

$$Y_{\text{Motion,Symptomatic},h} = \mu + b_{\text{Motion}} + m_{\text{Motion}} + \varepsilon_{\text{Motion},h} \quad (3.2)$$

For the identification of between sessions differences in performance equations 4.1 to 4.3 are used. Each equation refers to a specific participant group.

$$\text{Group M-NM} \quad \Delta(Y_h) = Y_{\text{Static},2,h} - Y_{\text{Motion},1,h} \quad (4.1)$$

$$\text{Group NM-M} \quad \Delta(Y_h) = Y_{\text{Motion},2,h} - Y_{\text{Static},1,h} \quad (4.2)$$

$$\text{Group NM-NM} \quad \Delta(Y_h) = Y_{\text{Static},2,h} - Y_{\text{Static},1,h} \quad (4.3)$$

where:

Y_{ijh} = performance of participant h in experimental session j under treatment i

i = treatment, where $i = \{\text{Motion, Static}\}$

C. VARIABLES

Beyond collecting demographic and sleep information, our investigation was based on the following variables.

- Independent Variables
 - Motion (versus no motion)
 - experimental session (first, second)
 - ten-minute block rank (first, second, etc.)
- Intermediate Variables
 - motion sickness related
 - subjective

- MSAQ Total: focusing on motion sickness in general
- MSAQ S (sopite) and SSS: focusing on sopite syndrome
- psychophysiological (objective)
 - electro-dermal activity (EDA): skin conductance mean (SC M) in μS
 - electrocardiography (ECG): Heart rate variability
 - electrogastrography (EGG): average power, and the percentage of EGG power, in the tachygastric frequency range (>4 cpm)
- Dependent Variables
 - SYNWIN composite and task scores (memory, arithmetic, visual detection, auditory detection)

One comment should be made the metrics we used or the detection of motion sickness. The reason for the decision to use both subjective and objective metrics is to increase the accuracy of motion sickness diagnosis (Stout & Cowings, 1993); it is preferable to use the combination of both these types of measures to either one alone.

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IV. ANALYSIS AND RESULTS

A. ANALYTICAL PLAN

The analytical plan is described in Figure 9 below. Initially, the statistical equivalence between the three participant groups was assessed (Box 1 in Figure 9). Then, the main scope of this study, the assessment of the effect of motion sickness on cognitive multitasking performance, was studied. The first part of these tests assessed the effect of order (Box 2). Then, the effect of motion sickness on changes in performance between sessions was assessed (Box 3). The flow of the analysis is depicted in Figure 9.

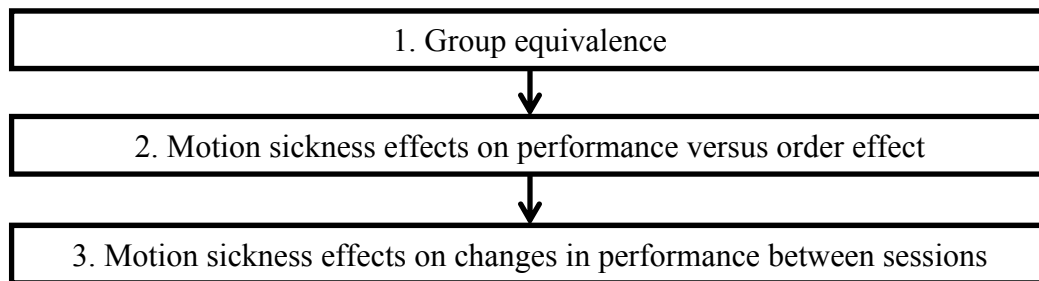


Figure 9. Data analysis flowchart.

Analysis of the effect of motion sickness on performance determined that motion sickness affected performance differently for the two sessions (Box 2). Participants who developed motion sickness symptoms in the second experimental session had degraded performance compared to those participants without symptoms. This phenomenon was not observed in the first session. This interaction between order and performance made the analysis more complex. The data could not be analyzed in the typical counterbalanced manner because of this interaction (Bordens & Abbott, 2006a, p.278–279). Instead, the decision was made to analyze the data independently in each experimental session.

Two methods were used: a correlational analysis between performance scores, and motion sickness severity, and a comparison of the Symptomatic and the Asymptomatic participants' scores in motion conditions. The basis of participants' classification into the two motion sickness groups (Symptomatic, Asymptomatic) was to

compare each participant's average symptom severity for both the motion and static conditions. If symptom severity in motion was greater than in static condition, the participant was classified as "Symptomatic." If motion sickness severity was less than or equal to the static condition, the participant was classified as "Neutral." Participants without symptoms in both static and motion conditions were classified as "Asymptomatic." This scheme can classify individuals who participate in both motion and static conditions. In the first motion session (M-NM group, n=20) there were ten Symptomatic, two Neutral, and eight Asymptomatic participants. In the second motion session (NM-M group, n=19) there were 11 Symptomatic, one Neutral, and seven Asymptomatic participants.

The effect of motion sickness on between sessions performance changes (Box 3) was assessed using repeated measures ANOVA with a between-subjects factor, participant's grouping. Three groups were used for this analysis, two in motion condition (Symptomatic and Asymptomatic), and one in the static conditions (i.e., the control group; participants in this group may report some mild symptoms but are, by definition, not "motion sick").

Microsoft Office Excel 2007 was used to develop the initial study databases. Analyses were conducted with JMP® Pro 9.0.0 by SAS Institute. Data normality was evaluated using the Shapiro-Wilk W test (Shapiro & Wilk, 1965). Parametric and nonparametric approaches were used accordingly for statistical analyses. To facilitate a better understanding of results in tables with multiple comparisons, $p < 0.05$ was indicated with a double asterisk "**," whereas a single asterisk "*" indicated p-values between 0.05 and 0.10. In the repeated measures ANOVA, sphericity was tested with Mauchly's test (1940). When appropriate, degrees of freedom were corrected using Greenhouse-Geisser (1959) estimates of sphericity.

Analysis of the effects of motion sickness on performance was based on average values per experimental session, without focusing on the temporal variations (Simon, 1976, p.13). Performance improvement of composite score focused on between sessions analysis based on ten-minute blocks as well as average value per session.

From a methodological perspective, the analyses of this study are based on the intermediate variable of motion sickness severity. The truly independent variable, motion, cannot be used for our purposes. The focus of our investigation is the development of motion sickness, which is the emergent outcome of the potentially nauseogenic motion. Therefore, the *ad hoc* “independent” variable in this work is motion sickness severity, which leads to the corresponding grouping of participants as Symptomatic or Asymptomatic based on whether they develop motion sickness symptoms or not.

B. DEMOGRAPHICS

The experimental methodology was the same in all data collection phases. The only difference was the post-test questionnaire administered at the end of participation (end of second ES) during the 2011 and 2012 data collection. Given that this change is a minor difference, we evaluated the demographic attributes of the combined population of participants.

Overall, 51 healthy, non-smoking, individuals participated in the experiment (45 males and 6 females, Air Force=4, Army=7, Navy=33, USMC=1, Civilian=4, NOAA=1, Other=1). Military ranks ranged from O2 to O5 (O2=5, O3=22, O4=18, O5=2). Participants were U.S. (n=24) and foreign citizens (24 Greeks, one from Brazil, one from Bahrain, and one from Norway).

A one-way between subjects ANOVA confirmed that the participant groups were homogeneous in their demographic information (age, gender, height, weight, morningness-eveningness (ME) tendency, MSSQ ratings, NEO personality traits, time between experimental sessions [inter-session interval], and the time of day the experimental session started). This analysis failed to reject the null hypothesis that the participant groups were statistically equivalent, Wilcoxon rank sum test, $p > 0.05$. However, it should be noted, especially with our small sample size, that non-rejection of the null does not confirm the null; rather, it merely shows that there is not sufficient evidence to reject the null. Participants’ basic demographic information is depicted in Table 3. The reported group differences are based on the Tukey post-hoc test (Tukey, 1953) when $p < 0.05$.

Table 3. Demographics

Parameter	All n=51 M (SD)	Group M-NM n=20 M (SD)	Group NM-M n=19 M (SD)	Group NM-NM n=12 M (SD)
Age (years)	35.4 (5.74)	34.8 (5.00)	35.6 (7.05)	36.1 (4.89)
BMI	26.7 (3.52)	27.1 (3.94)	26.4 (3.82)	26.4 (2.29)
Daily sleep need (hrs)	7.18 (1.02)	7.55 (0.724)	6.70 (1.25) †	7.33 (0.784)
Reported daily sleep (hrs)	7.06 (1.13)	7.33 (1.21)	6.87 (1.14)	6.90 (0.968)
ME tendency score	53.5 (8.95)	52.5 (8.61)	55.4 (11.1)	52.3 (5.08)
NEO				
N	15.6 (7.58)	18.6 (7.72)	14.0 (6.88)	13.0 (7.26)
E	29.6 (5.53)	29.2 (5.80)	30.5 (5.49)	28.9 (5.40)
O	27.8 (5.72)	28.4 (7.13)	27.4 (4.68)	27.4 (4.89)
A	30.9 (4.83)	29.6 (5.40)	32.5 (4.46)	30.4 (3.87)
C	33.9 (6.46)	34.3 (8.35)	33.3 (4.89)	34.1 (5.42)
MSSQ	15.3 (24.7)	14.4 (20.3)	20.1 (34.2)	9.26 (8.38)

† Differences between groups NM-M and M-NM

Although all three groups were equivalent in MSSQ scores, group NM-M reported greater susceptibility to motion sickness. This issue is associated primarily with Participant 12 whose MSSQ score was 146. Without Participant 12, group NM-M had an average MSSQ of 13.1 (SD=15.5, MD=9.68). The MSSQ average of our population is much lower than the 50th percentile (reached at approximately MSSQ=40). These results suggest that their average susceptibility was less than that of a normal population.

Based on their ME score, participants were grouped as moderately evening type (n=4, 7.84%), neither type (n=28, 54.9%), moderately morning type (n=17, 33.3%), and definitely morning type (n=2, 3.92%). The actual inter-session interval was 6.61 days (SD=1.28, MD=7).

C. EQUIVALENCE OF PARTICIPANT GROUPS

Initially, the statistical equivalence of the three participant groups (M-NM, NM-M, NM-NM) was assessed at the beginning of the study for SYNWIN composite score. Analysis of variance was based on the average values during the first two 10-minute blocks per participant. The ANOVA identified differences between the three groups for composite score ($F(2,48) = 2.76$, $p = 0.074$). Multiple comparisons using the Tukey post-hoc test showed a significant difference ($p=0.090$) between the NM-M ($M=997$, $SD=204$) and NM-NM group ($M=1179$, $SD=261$). This analysis of SYNWIN composite scores shows that M-NM and NM-M, as well as M-NM and NM-NM, are statistically equivalent in the beginning of the study. An attempt is made to address the issue of non-equivalence between groups NM-M and NM-NM in the beginning of the study with the normalized performance values.

However, this approach introduces the question whether the absolute level of performance in the beginning may influence the degree of performance improvement later in the study. Further analysis of normalized data showed that, compared to the beginning (blocks 1 and 2), all three participant groups (M-NM, NM-M, NM-NM) had statistically equivalent percentage-wise performance improvement at the end of the first session (block 6), the beginning of the second session (blocks 7 and 8), and the at the end of the second session (block 12) (one-way ANOVA, $p > 0.30$). These results suggest that the absolute values of performance cannot be used for comparisons between groups.

In an attempt to overcome this issue (Bordens & Abbott, 2006a, p. 112), SYNWIN performance scores were transformed by a normalization method, independently for each participant. Two forms of the normalized scores were used. For each participant, the intra-session score (henceforth referred to as “INTRA”) was calculated over each ten-minute block using the following equation: INTRA metric of the k -th ten-minute block of the i -th experimental session = absolute value of the metric divided by the average value of the metric in the first two ten-minute blocks of the corresponding i -th experimental session. The inter-session form (henceforth referred to as “INTER”) was the same as the INTRA form, but the normalization was performed using the first two ten-minute blocks of the first session as the baseline for normalization. In

both normalization cases, the baseline includes both the first two ten-minute blocks to minimize regression toward the mean (Kirk, 2009), i.e., a phenomenon where a variable is extreme on its first measurement and tends to be closer to the average on subsequent measurements. This approach goes beyond the absolute level of performance. Therefore, this analysis will focus on relative (normalized) values of SYNWIN performance.

Figure 10 provides an aggregate depiction of how the SYNWIN composite score developed over time. Diagrams with a detailed depiction of composite score in each 10-minute block are included in the appendix.

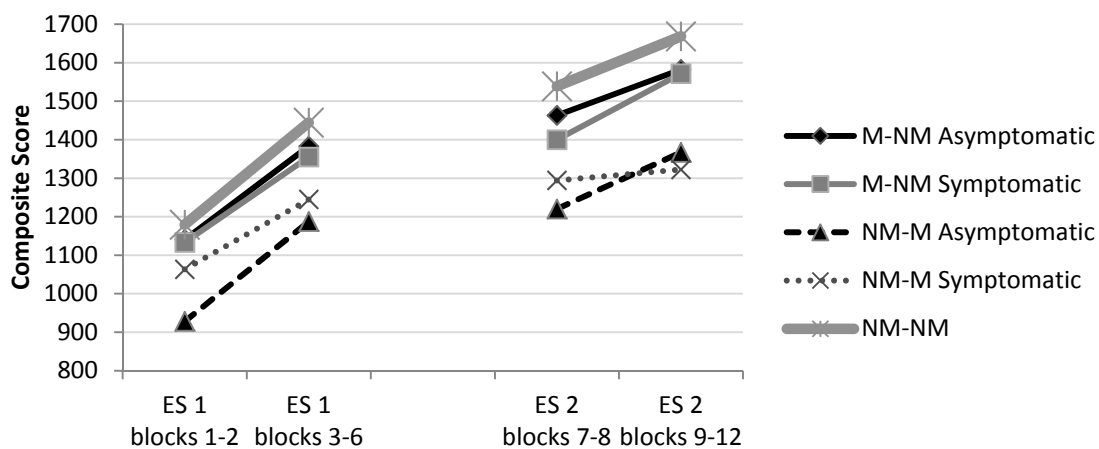


Figure 10. Development of composite score.

D. MOTION SICKNESS SEVERITY BETWEEN EXPERIMENTAL SESSIONS

This section explores how motion sickness severity changed between sessions. A paired t-test of MSAQ Total between ES 1 and ES 2 showed that symptom severity decreased from ES 1 to ES 2 for group M-NM (ES 1: $M=14.5$, $SD=5.29$; ES 2: $M=11.5$, $SD=1.07$; $t(19)=-0.87$, $p=0.005$), increased for group NM-M (ES 1: $M=11.3$, $SD=0.395$; ES 2: $M=14.3$, $SD=7.46$; $t(18)=1.77$, $p=0.047$), and remained the same for group NM-NM (ES 1: $M=11.7$, $SD=0.929$; ES 2: $M=11.6$, $SD=0.669$; $t(11)=-0.899$, $p=0.194$).

In motion conditions all 16 symptoms were reported, whereas only six symptoms were reported in the static condition. Table 4 shows how many participants reported each

of the 16 symptoms included in the MSAQ. For each symptom the letter in parentheses denotes the cluster to which the symptoms belong (gastrointestinal—G, peripheral—P, central—C, sopite—S).

Table 4. Number of participants reporting the MSAQ symptoms.

Symptom I feel:	Participant group					
	M-NM (n=20)		NM-M (n=19)		NM-NM (n=12)	
	ES 1 (motion)	ES 2 (static)	ES 1 (static)	ES 2 (motion)	ES 1 (static)	ES 2 (static)
Faint-like (C)	2	0	0	1	0	0
Like I was spinning (C)	2	0	0	1	0	0
As if I may vomit (G)	3	0	0	1	0	0
Lightheaded (C)	4	1	0	2	0	0
Drowsy (S)	3	0	0	2	0	0
Clammy/ cold sweat (P)	4	0	0	2	0	0
Sick to my stomach (G)	3	0	0	5	0	0
Disoriented (C)	5	0	0	3	0	0
Dizzy (C)	5	0	0	3	0	0
Nauseated (G)	5	0	0	5	0	0
Annoyed/ irritated (S)	8	2	0	3	0	0
Sweaty (P)	7	2	0	5	0	0
Hot/ warm (P)	7	2	1	5	2	1
Queasy (G)	5	0	0	8	0	0
Tired/ fatigued (S)	9	3	4	6	2	2
Uneasy (S)	9	0	0	6	1	0

To assess classification changes (Asymptomatic, Neutral, Symptomatic) between all three participant groups, a modified classification method was used. For each participant, we compared the average symptom severity between ES 1 and ES 2. If symptom severity in ES 2 was greater than in ES 1, the participant was classified as “Symptomatic.” If motion sickness severity was less than or equal to ES 1, the participant was classified as “Neutral.” Participants without symptoms in both ES 1 and ES 2 were classified as “Asymptomatic.” Results show that symptom severity increases in motion conditions compared to static conditions. No differences in symptom severity were identified between sessions in the control group (NM-NM). Table 5 shows these results.

Table 5. Modified symptom classification between participant groups.

Modified Group	Participant group					
	M-NM		NM-M		NM-NM	
	From ES 1 to ES 2	From ES 2 to ES 1	From ES 1 to ES 2	From ES 2 to ES 1	From ES 1 to ES 2	From ES 2 to ES 1
Asymptomatic	8	8	7	7	7	7
Neutral	11	2	1	11	4	2
Symptomatic	1	10	11	1	1	3

E. MOTION SICKNESS, PERFORMANCE, AND THE DIFFERENTIAL EFFECT OF SESSION

This section explores two issues (Step 2 in Figure 9); the effect of motion sickness and sopite syndrome symptoms on SYNWIN scores, and the order effect associated with the experimental sessions (ES). The analysis focuses on motion conditions with groups M-NM and NM-M.

First, a correlational analysis was performed between SYNWIN scores (INTRA) and the subjective metrics of motion sickness (MSAQ indices, and SSS) in motion conditions. All values were averaged per participant and motion ES. Correlational analysis was based on Spearman's rank order correlation coefficient (Spearman's rho).

The composite score showed a decrement only in motion ES 2 for MSAQ Total (rho= -0.726, $p < 0.001$), MSAQ S (rho= -0.606, $p = 0.006$), and SSS (rho=-0.472, $p = 0.042$). The memory task scores decreased for MSAQ Total in ES 2 (rho= -0.545, $p = 0.016$), MSAQ S (rho=-0.475, $p = 0.040$), and SSS in ES 2 (rho= -0.483, $p = 0.036$). Arithmetic task scores decreased for MSAQ Total in ES 2 (rho= -0.600, $p = 0.007$), and for MSAQ S in ES 2 (rho= -0.481, $p = 0.037$). No significant associations were identified for the visual and auditory tasks. Table 6 integrates these findings.

Table 6. Associations between SYNWIN performance scores (INTRA) and motion sickness severity.

Scores	ES 1 in motion Group M-NM	ES 2 in motion Group NM-M
Composite	-	MSAQ Total, MSAQ S, SSS
Memory task	-	MSAQ Total, MSAQ S, SSS
Arithmetic task	-	MSAQ Total, MSAQ S
Visual task	-	-
Auditory task	-	-

No significant correlations were identified in the static conditions. Next, we assessed whether Symptomatic participants in motion conditions demonstrated reduced performance when compared to Asymptomatic participants. Although motion sickness severity was not different between the two motion sessions (Wilcoxon Rank Sum test, $X^2(1)=0.144$, $p=0.705$), our analysis showed significant score differences between Symptomatic and Asymptomatic participants only in the second experimental session while in the motion conditions. These findings are demonstrated in Table 7 and further depicted in Figure 11.

Table 7. SYNWIN (INTRA) score differences between Symptomatic/Asymptomatic individuals by experimental session (motion condition).

Scores	ES (motion)	Asymptomatic M% (SD%)	Symptomatic M% (SD%)	Significance (Wilcoxon Rank Sum test)	Cohen's d
Composite	1	123 (12.7)	122 (14.2)	$X^2(1)=0.097$, $p=0.755$	-
	2	113 (6.17)	103 (10.1)	$X^2(1)=6.47$, $p=0.011^{**}$	1.20
Memory	1	141 (33.8)	172 (142)	$X^2(1)=0.387$, $p=0.534$	0.300
	2	138 (52.2)	105 (12.5)	$X^2(1)=3.81$, $p=0.050^{**}$	0.870
Arithmetic	1	139 (27.9)	134 (33.6)	$X^2(1)=0.008$, $p=0.929$	0.162
	2	117 (11.4)	102 (22.5)	$X^2(1)=5.33$, $p=0.021^{**}$	0.841
Visual	1	96.3 (4.03)	97.1 (4.28)	$X^2(1)=0.244$, $p=0.626$	0.195
	2	98.5 (2.94)	97.9 (2.70)	$X^2(1)=0.033$, $p=0.855$	0.213
Auditory	1	114 (24.6)	118 (24.4)	$X^2(1)=0.641$, $p=0.423$	0.163
	2	108 (11.5)	106 (11.5)	$X^2(1)=0.741$, $p=0.389$	0.174

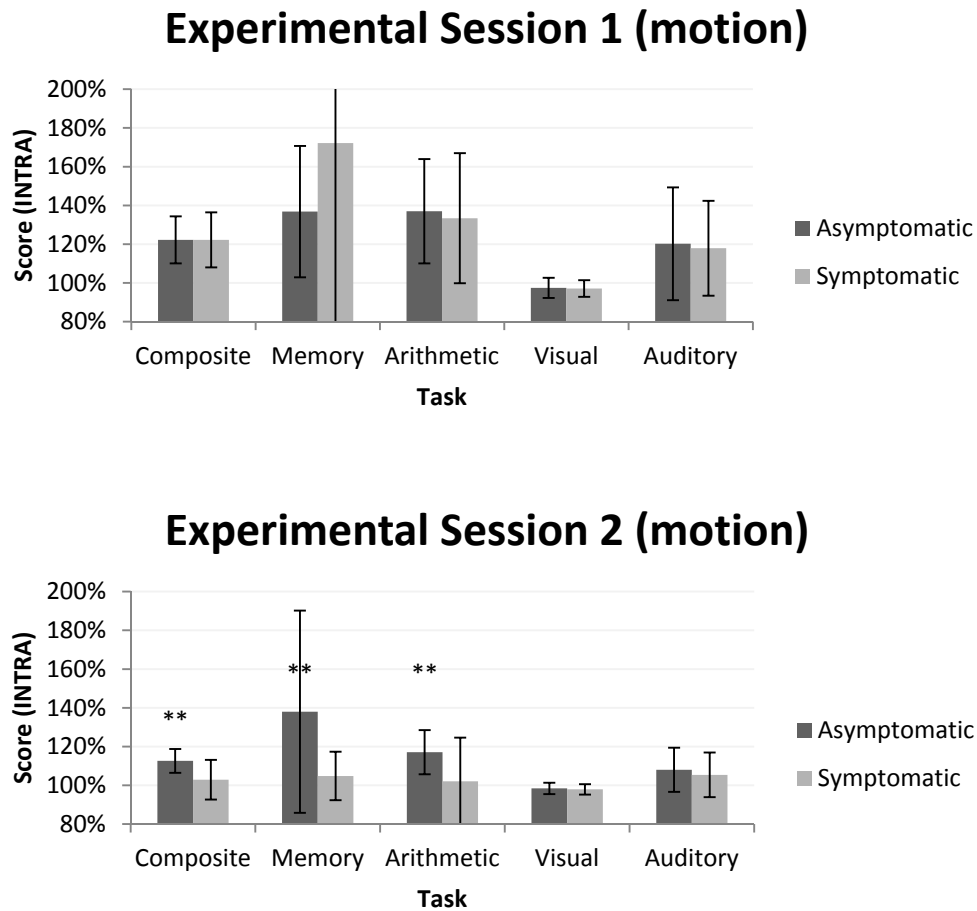


Figure 11. SYNWIN score (INTRA) differences between Symptomatic and Asymptomatic individuals by experimental session (motion condition).

These results show that differences in performance between Symptomatic and Asymptomatic participants in motion were significant only in the second motion session. Significant differences were identified for the composite scores, as well as for the memory and arithmetic tasks. Overall, these findings show a consistent pattern. During the first motion session, participants seem to overcome mild motion sickness and sopite syndrome symptoms, whereas during the second motion session, motion sickness symptoms take a toll on performance. In the second motion session, the reductions in mean performance were 9.43% for the composite score, 31.7% for the memory task, and 14.7% for the arithmetic task. These results are further emphasized by the fact, that in static conditions, performance was not associated with symptoms.

We repeated the correlational analysis, this time between SYNWIN performance (INTRA) and psychophysiological metrics. In the first session, the memory task score was correlated with SC ($\rho = 0.424$, $p = 0.063$). In the second session, the arithmetic task score was correlated with the percentage of EGG power in the tachygastic frequency range (>4 cpm) ($\rho = 0.437$, $p = 0.062$), and the visual task score was correlated with SC ($\rho = 0.447$, $p = 0.055$). These results are sporadic and do not constitute a pattern; therefore, they should be considered inconclusive indication of the association between performance and psychophysiological metrics of motion sickness.

F. THE EFFECT OF MILD MOTION SICKNESS ON PERFORMANCE DIFFERENCES BETWEEN EXPERIMENTAL SESSIONS

This section assesses between-session SYNWIN performance differences (Step 3 in Figure 9). Analysis addressed the differences in performance between experimental sessions in association with the presentation of motion and the development of motion sickness symptoms. First, analysis was focused on performance changes from the first ES (block 6) to the beginning of the second ES (block 7). A within-subject ANOVA between blocks 6 and 7 with a between-subjects factor (five participant groups: Symptomatic in M-NM group, Asymptomatic in M-NM group, Symptomatic in NM-M group, Asymptomatic in NM-M group, NM-NM group) did not identify performance changes across time ($F(1, 44) = 1.40$, $p = 0.243$), or differences between groups (interaction of ES and participant group: $F(4, 44) = 0.863$, $p = 0.494$). Hence, for all groups, performance did not change at the beginning of ES 2 compared to the end of ES 1.

Then, analysis was focused on performance improvement from the first ES to the second ES (blocks 9 to 12). A one-way repeated measures ANOVA showed a significant interaction between session and group in composite score, $F(4, 44) = 2.94$, $p = 0.031$. This interaction was identified between Symptomatic participants in group NM-M and participants in group NM-NM, $F(1, 21) = 6.67$, $p = 0.017$, but not among the rest of the groups ($p > 0.500$). These results show that, excluding Symptomatic participants in group NM-M in the second experimental session, all participant groups showed a comparable increase in performance.

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V. DISCUSSION

In this final chapter, we integrate the picture shown by these findings. First, we focus on the experimental results, the “what we found.” We discuss the effect of mild motion sickness and sopite syndrome on cognitive multitasking performance, as influenced by experimental session. Then we address performance improvement between sessions. Second, we investigate and postulate “why” we found these results. We assess motion sickness from a stress perspective and propose an explanation about performance deterioration based on attentional resources and executive functioning. The discussion concludes by proposing an improved definition of sopite syndrome.

A. MOTION SICKNESS, PERFORMANCE, AND THE DIFFERENTIAL EFFECT OF SESSION

The nauseogenic motion stimulus included the superposition of three independent 0.167 Hz sinusoidal motions. In the z-axis, it was a +/- 2 inches displacement (heave). In the y and x axes, it was a +/- 15 degrees roll and pitch. On average, the severity of motion sickness in our study was mild. Although normative data for the subjective MSAQ scale do not exist, the maximum severity of symptoms assessed by MSAQ Total in our study was approximately 43 with the maximum rating of the scale being 100. From a qualitative perspective, the average severity of motion sickness developed by the Symptomatic participants is similar to that defined as “moderate malaise MIIA” (E. F. Miller & Graybiel, 1974).

Overall, the results show that cognitive multitasking performance deteriorated with the development of mild motion sickness and soporific symptoms. The results also provide evidence for an order effect. There is a consistent pattern with motion sickness and soporific symptoms demonstrating a pronounced association with performance only in the second experimental session. Performance differences in composite scores (9.43%), as well as in the memory (31.7%) and arithmetic task scores (14.7%), between Symptomatic and Asymptomatic participants were significant, but only in the second experimental session, not the first.

We postulate that the differential effect of session in the association between symptomatology and multitasking performance may be related to the attentional resources allocated to performing the multi-task. This hypothesis is based on the fact the two experimental sessions differ only in the degree of skill that the participants have acquired in performing the multi-task. The framework for this hypothesis is discussed later.

Overall, we conclude that multitasking cognitive performance can deteriorate even in motion environments where motion sickness and sopite syndrome symptoms are not severe. The basic findings of our experiment are diagrammatically conceptualized in the Figure 12.

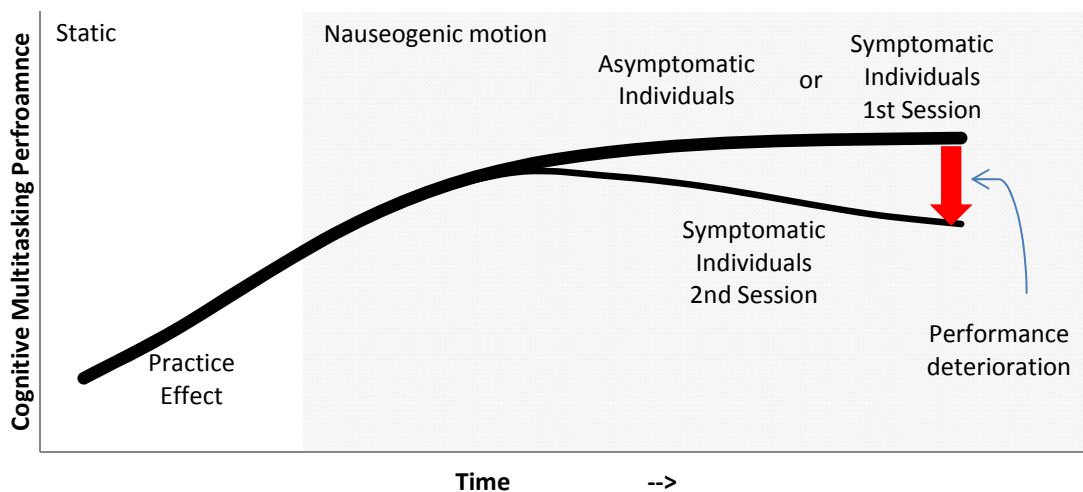


Figure 12. Conceptual depiction of multitasking performance versus nauseogenic motion stimulus, motion sickness severity, and experimental session.

The previous figure depicts the initial improvement of performance when initially learning the multitasking environment. The initial acquisition is followed by a plateau representing a stabilization of performance over time. Obviously, this is a simplification, but it is an accurate depiction of the results of our experiment. The introduction of potentially nauseogenic motion further complicates the individual's performance. Excluding biodynamic effects, an individual's performance is confounded by the severity of motion sickness and sopite syndrome symptomatology. Focusing on Symptomatic

individuals, and assuming a continuous nauseogenic stressor, the associated symptomatology will gradually become more severe. In the second experimental session, after an initial period of non-significant effects, performance will demonstrate a more rapid deterioration. On the other hand, the performance of Asymptomatic individuals does not seem to be affected by mild motion *per se*. From a methodological perspective, this result means that the performance deterioration associated with motion sickness should be assessed as the performance difference between Symptomatic and Asymptomatic individuals performing under the same motion conditions. This observation is congruent with Reason and Brand's (1975, p. 55) comment that some early motion research suffered from the lack of a control group of "non-sick passengers." Many studies, however, evaluate performance degradation due to motion sickness by comparing performance between motion and static conditions with the same or different individuals.

B. THE EFFECT OF MILD MOTION SICKNESS ON PERFORMANCE DIFFERENCES BETWEEN EXPERIMENTAL SESSIONS

Results showed that performance did not change in the beginning of ES 2 compared to the end of ES 1. The pattern of performance retention was not associated with the existence of motion stimulus or the development of mild motion sickness symptoms in the first session. Therefore, our results suggest that mild motion sickness does not interfere with performance retention in a novel cognitive multitasking environment. A probable explanation for this finding may be found in the level at which mild motion sickness interferes with cognitive performance in the first experimental session, where participants are still novices. As we demonstrated earlier, our first session results show that mild motion sickness does not degrade performance. Probably, participants overcome the detrimental effects of mild motion sickness by focusing on the multi-task, and practice leads to the development of skills. Over time, the acquired skills lead to increased performance (Proctor & Wang, 1998).

It would be interesting to assess performance retention between the second and a third session. In this case, participants would not be novices. Given that motion sickness led to deterioration in performance in the second session, should we expect an interaction of motion sickness and skill acquisition between subsequent sessions (e.g., from second to third)?

C. MOTION SICKNESS AS A STRESSOR

Considering motion sickness as a stressor, our findings may be explained from a perspective of performance under stress. The deterioration of task performance in cognitive tasks (memory and arithmetic) is congruent with research on the effects of stress on performance (van Hiel & Mervielde, 2007). Simple tasks needing automated responses will suffer less from stress than responses in complex tasks with underlying cognitive control (Yerkes & DoDson, 1908). In the SYNWIN multitasking battery, a reasonable ordering of the four tasks based on the resources needed would put the arithmetic task first, followed by the memory task. The arithmetic task can be identified as “resource limited” in the sense that mathematical reasoning probably demands the association of resources to obtain maximum performance (Wickens, 2002). The visual and auditory tasks in the multitasking battery seem to be last in this list. Their nature (lack of visual search, easily identifiable signals) locate them closer to being automated, in the sense that they need a minimal amount of resources (Fitts & Posner, 1967).

Why, then, do we observe this deleterious effect of motion sickness? Is it due to motivation (or lack thereof), or because of changes in resource capacity, such as limitations on working memory? Future research efforts can explore this issue in greater detail.

From an attentional capacity overload perspective (Matthews & Desmond, 1995), our experimental results seem reasonable. The arithmetic task suffered the most, followed by the short-memory task. The visual and auditory tasks did not seem to be affected. This hierarchy is in congruence with existing literature on multiple resource theory (Wickens, 2002; Wickens & Hollands, 2000). This theory postulates that the sensory processing of the peripheral visual and auditory systems is relatively resource-free (Wickens and Hollands, 2000). In this case, the denial of use of attentional resources resulting from

motion sickness will have a small effect on the visual and auditory tasks. Our results suggest that motion sickness acts like a distraction or a diversion, and, therefore, that difficulties in concentration should be considered among the major symptoms in mild motion sickness. As already noted, however, research also has identified that being involved with a mental task may decrease motion sickness severity (Bos, 2011; Correia & Guedry, 1966; Graybiel, 1968). The antagonistic association between motion sickness severity and cognitive effort may be explained from a cognitive resources and cognition control perspective.

Consider the dichotomy of executive versus automatic control on cognition (Ackerman, 1987, 1988; Anderson, 1996). Executive control, executive function, or control processing is an endogenous control and coordination of neurocognitive processes to attain a novel or complex goal (Logan, 1985; Schneider & Shiffrin, 1977). Automatic control is evident in learned, or automatic responses (Norman & Shallice, 1986; Schneider & Shiffrin, 1977). As already noted, our participants had a single goal, to maximize the composite score of the multitasking battery. Over time, they developed a strategy to perform the four tasks concurrently. This process reflects an implementation of an executive control of their cognitive resources.

During the first one-hour session, participants developed and started to implement their cognitive strategy. Accumulation of practice by repetition leads to more efficient multitasking performance, and to increasingly more automated responses. Research has shown that as skills develop, brain regions not necessary for task performance become less active (Smith, McEvoy, & Gevins, 1999). This finding is consistent with earlier research identifying the differences in attentional capacity allocation between novices and experts (Fitts, 1964; Fitts & Posner, 1967). Automatic control progressively replaces executive control. Therefore, cognitive resources formerly being allocated to the multitasking battery are, to some extent, released.

This process starts in the first session, but is more evident during the second. We postulate that these released resources are partially diverted to monitoring the malaise associated with motion sickness, probably through a control structure like the central executive proposed by Baddeley (1986) or Norman and Shallice's Supervisory

Attentional System (SAS) (Norman & Shallice, 1986; Shallice, 1988). Therefore, motion sickness acts as a distractor by withholding or denying the use of these attentional resources. In the absence of motion sickness, or in the existence of mild malaise, a task may be performed in the non-overloading zone of available attentional resources. By limiting available capacity, the existence of motion sickness “pushes” the task into the near- or overloaded zone. The point where motion sickness starts to cause significant interference seems to be associated with the executive function noted earlier. It is interesting that Wickens (2002) has identified that multiple resource theory does not address in an adequate manner resource allocation or “engagement”:

[There] are circumstances in which one task demands or attracts so much attention to itself that any benefits that might otherwise have been realized by its separate resources are eliminated, as full attention is given to that task; as a consequence, the concurrent task is essentially “dropped” altogether (Wickens, 2002, p. 173).

He also notes an example for this situation in which cellular phone conversations were so “engaging” that drivers totally neglected aspects of the concurrent driving task, even though the two tasks were quite (but not totally) separate in their resource demands (Strayer & Johnston, 2001; Wickens, 2002).

The association between executive control and motion sickness extends to the individual’s motivation. A novel task may be more interesting and alerting. In this case, an individual is more self-motivated to perform a novel task. From an executive function perspective, this increased motivation reflects the allocation of cognitive resources to the task, a process that is reversed when motivation decreases. We believe that this theoretical scheme provides a plausible explanation for why motion sickness’s effects on performance are associated with motivation, task involvement, and task novelty.

Furthermore, it is consistent with findings of other researchers. Dobie and his colleagues showed that encouragement to suppress symptoms (“cognitive counseling”) increased tolerance (Dobie et al., 1987; Dobie et al., 1989), whereas anecdotal data and subjective reports since WWII suggest that even sick individuals can continue performing acceptably if they are highly motivated (Baker, 1966; Birren, 1949; Greenberg, 1946; Tyler & Bard, 1949). Griffin (1990) noted that “it is variously reported that mental

activity is beneficial in minimizing sickness,” a comment supported by research (Bos, 2011; Correia & Guedry, 1966). Alexander and colleagues approached these issues by differentiating “peak” from “maintenance efficiency.” Peak efficiency refers to exertion that sick individuals demonstrate when emergency performance is needed, whereas maintenance efficiency refers to daily routine (Alexander et al., 1945d). Results suggest an inverse relationship between motion sickness effects on performance and the cognitive effort focused on performing a task.

The conceptual framework described above does not contradict the neural mismatch basis of motion sickness, but merely includes it as an integral part. The neural mismatch error signal is the causal factor for the severity of motion sickness. Therefore, we expect that the attentional resources diverted to motion sickness distraction are, to some extent, related to the severity of this causal error. When the error starts to decrease, either because of adaptation or a reduction in the nauseogenic motion stimulus, there is a corresponding reduction of the distractive malaise. In this case, a decrease of motion sickness will be associated with reduced attention to this distraction, and cognitive resources will be released and allocated towards performing the assigned task.

So far, we have provided information regarding the background literature on the association between motion sickness and cognitive resources, postural control, sensory integration, and disorientation, and how motion sickness draws attention from cognitive activities. We hypothesize that our results combined with the previously described background can be integrated in the conceptual framework depicted in Figure 13.

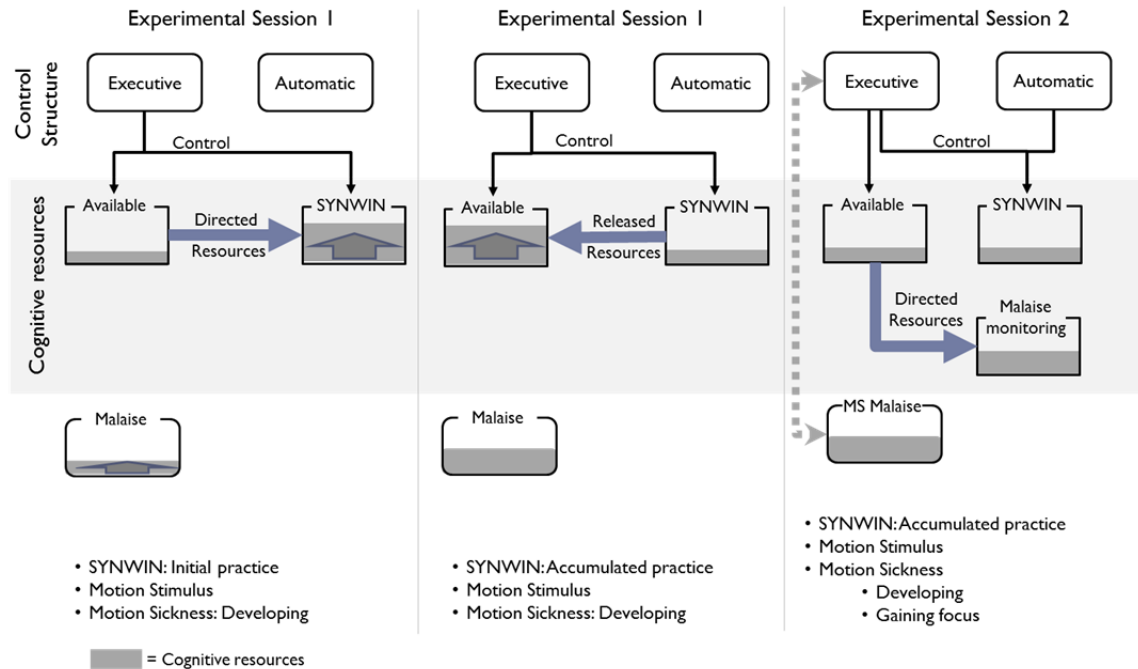


Figure 13. Hypothetical conceptualization of cognitive resources control versus motion sickness severity and experimental session.

This figure depicts the following hypothesis: initially, cognitive resources are directed towards performing the new task. Over time, practice leads to skill acquisition. This leads not only to a better performance, but also to a progressive release of cognitive resources earlier allocated to performing the task. Meanwhile, motion sickness malaise also increases. At some point, increased levels of malaise divert attention from the task to monitoring motion sickness.

D. AN APPROACH TO DEFINING SOPITE SYNDROME

In their seminal paper, Graybiel and Knepton (1976) described sopite syndrome as a symptom-complex centering around drowsiness. They noted that typical symptoms of the syndrome are yawning, drowsiness, disinclination for work, either physical or mental, and a lack of participation in group activities. This description was followed by the definition provided in ISO 5805:1997, “inordinate sleepiness, lassitude or drowsy inattention induced by vibration, low-frequency oscillatory motion (e.g., ship motion) or general travel stress...” (ISO, 1997, p. 11).

However, both definitions of sopite syndrome have limitations. The 1976 approach is based on the individuals' state (drowsy), without emphasizing that the symptoms associated with sopite syndrome can be observed even in the absence of the syndrome. However, Hill (1936) discussed the association between seasickness and drowsiness, apathy, or mental lethargy, emphasizing that these symptoms occur in the absence of actual somnolence. Similarly, Lawson and Mead (1998) indicated that sopite syndrome is distinct from the state of fatigue. The 1997 definition noted the "inordinate" attribute of sleepiness and lassitude, but restricted the definition of sopite syndrome to apply only to real motion. However, it is known that soporific symptomatology is evident even in a setting with apparent motion.

Based on these descriptions of sopite syndrome, we propose the following definition:

Sopite syndrome is a general term describing a symptom-complex centered on excessive drowsiness, lassitude, lethargy, and reduced ability to focus on an assigned task, in real or apparent motion settings, at levels that cannot be accounted for in a healthy individual by sleep deprivation and mental or physical fatigue due to increased activity.

This definition addresses the limitations of earlier approaches and provides an adequate conceptual framework for research.

E. EXTERNAL VALIDITY

Our results show that even mild motion sickness severity can have a detrimental effect on cognitive multitasking performance. These findings are obtained from a military population with no prior knowledge of the tasks involved. We used a head-mounted display to facilitate the appropriate environmental conditions resembling a closed moving compartment without visual cues from the external environment. These conditions are commonly found onboard ships or other moving platforms where personnel do not obtain sensory input from the external world.

Moreover, our results quantify the association between motion sickness and an order effect. In our experiment, the order effect is associated with learning and motivation to perform. It is reasonable to expect that highly motivated individuals will

further ameliorate the deleterious effects of motion sickness, even at severity levels higher than the ones observed in our study. However, this postulation is yet to be identified in a quantifiable manner.

We believe that a significant contribution of this study is the methodological approach to assess motion sickness effects among individuals performing in the same environment under the same skill acquisition level. Earlier studies generally compare the combined effect of motion and motion sickness against performance in static conditions. In this case, results are confounded by motion interference. The comparison of “sick” and “not sick” individuals under the same motion conditions not only delineates this problem, but it is also more valid operationally in the sense that we address specifically the problem of motion sickness rather than motion.

Our laboratory results are a step toward further exploration of motion sickness effects in complex operational environments. Research is needed with real-world tasks in the actual operational environment to verify the external validity of our findings.

F. CAVEATS

1. Randomization

During the 2010 and 2011 data collection phases, we assigned participants randomly to M-NM and NM-M groups, whereas all participants during the 2012 phase were included in the NM-NM group. Although all participants were from the same population, future replication of this study should incorporate a better randomization process.

2. Non-stabilized Performance

Participants in this experiment were initially novices. For some of them, performance was increasing in both experimental sessions. To address the effect of mild motion sickness, future efforts should use participants with stabilized performance

3. Statistical Equivalence of Participant Groups

Analysis showed that the three participant groups did not have statistically equivalent SYNWIN performance at the outset of the experiment. In an attempt to overcome this issue (Bordens & Abbott, 2006a, p. 112), the analytical approach included normalizing performance for each participant. However, future efforts should use statistically equivalent groups.

4. The Population used

Most of our participants were military officers and relatively young. This population led to restricted variability. The attributes of this group may not be representative of the general population.

5. The Multitasking Battery

SYNWIN is a useful multitasking battery including four simple tasks. Although the four tasks resemble a real operational multitasking environment, it still remains a simulation. Further research in operational conditions is needed to explore the external validity of the results.

6. Symptomatic Groups Classification

There is a long history of discussion about how and when an individual should be defined as suffering from motion sickness. As early as 1949, Birren stated that “every study of motion sickness has been confronted with the issue of deciding when a man is motion sick” (Birren, 1949). Reason and Brand noted more than 40 years ago, “...there is no single best way of assessing experimentally-induced motion sickness. The scheme adopted by any particular investigator will reflect his inclinations..., and will necessarily be governed by the nature of the investigation” (Reason & Brand, 1975, p. 82). Known measures of motion sickness share non-specificity as a common attribute (for example Lang, Sarna, & Shaker, 1999; Wiker & Pepper, 1978). From a methodological standpoint, the challenge is to classify participants based on their symptom severity scores in such a way to minimize “misreported” motion sickness, hence the development of minor symptoms, either because of reasons other than motion sickness, or in the

absence of a nauseogenic stimulus. The basis of the classification methodology used in this study was to compare each participant's average symptom severity between motion and static conditions or in the case of the NM-NM group, the difference between two experimental sessions. However, future efforts should explore alternative method to identify motion sick individuals.

VI. RECOMMENDATIONS FOR FUTURE RESEARCH

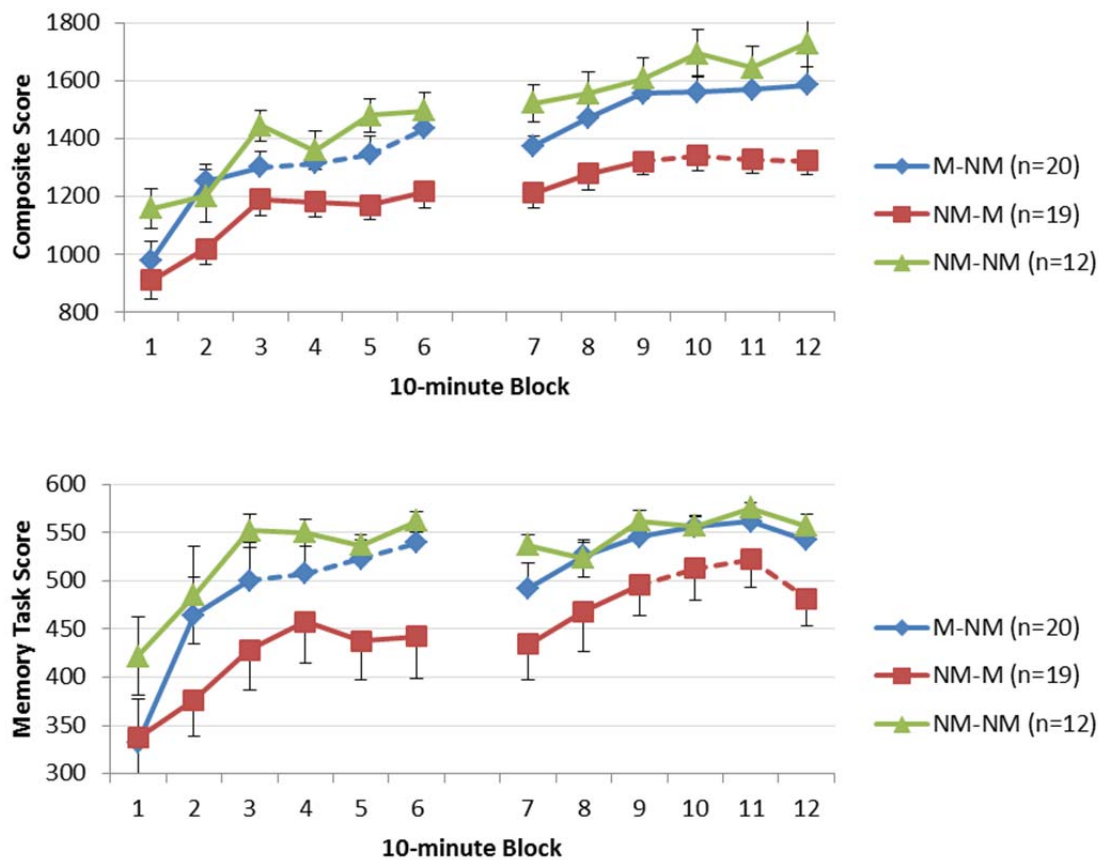
This work raises more questions than answers. Given that mild motion sickness and sopite syndrome are associated with performance deterioration, we should focus on the exploration of multitasking performance of adapted personnel in real operational environments. This exploration should expand in the following directions:

- Performance assessment in the absence of any motion sickness symptoms. Therefore, we would solely assess soporific effects;
- Performance during the adaptation process associated with sopite syndrome, a phenomenon yet to be investigated in detail (Graybiel et al., 1965; Graybiel & Knepton, 1976);
- The effect of intrinsic (personality traits) and extrinsic motivation (leadership) on personnel multitasking performance under nauseogenic conditions;
- The confounding effect of sleep, fatigue, or depression on the operational consequences of soporific effects, an issue already noted by some researchers (Lawson & Mead, 1998).

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APPENDIX

The graphs in Figure 14 depict the time development of the five SYNWIN scores, the composite, and the four task scores. Motion was presented to group M-NM during blocks 3 to 6, whereas group NM-M received motion during blocks 9 to 12. Vertical bars refer to Standard Error of the Mean (SEM). Data points connected by dotted lines refer to motion conditions.



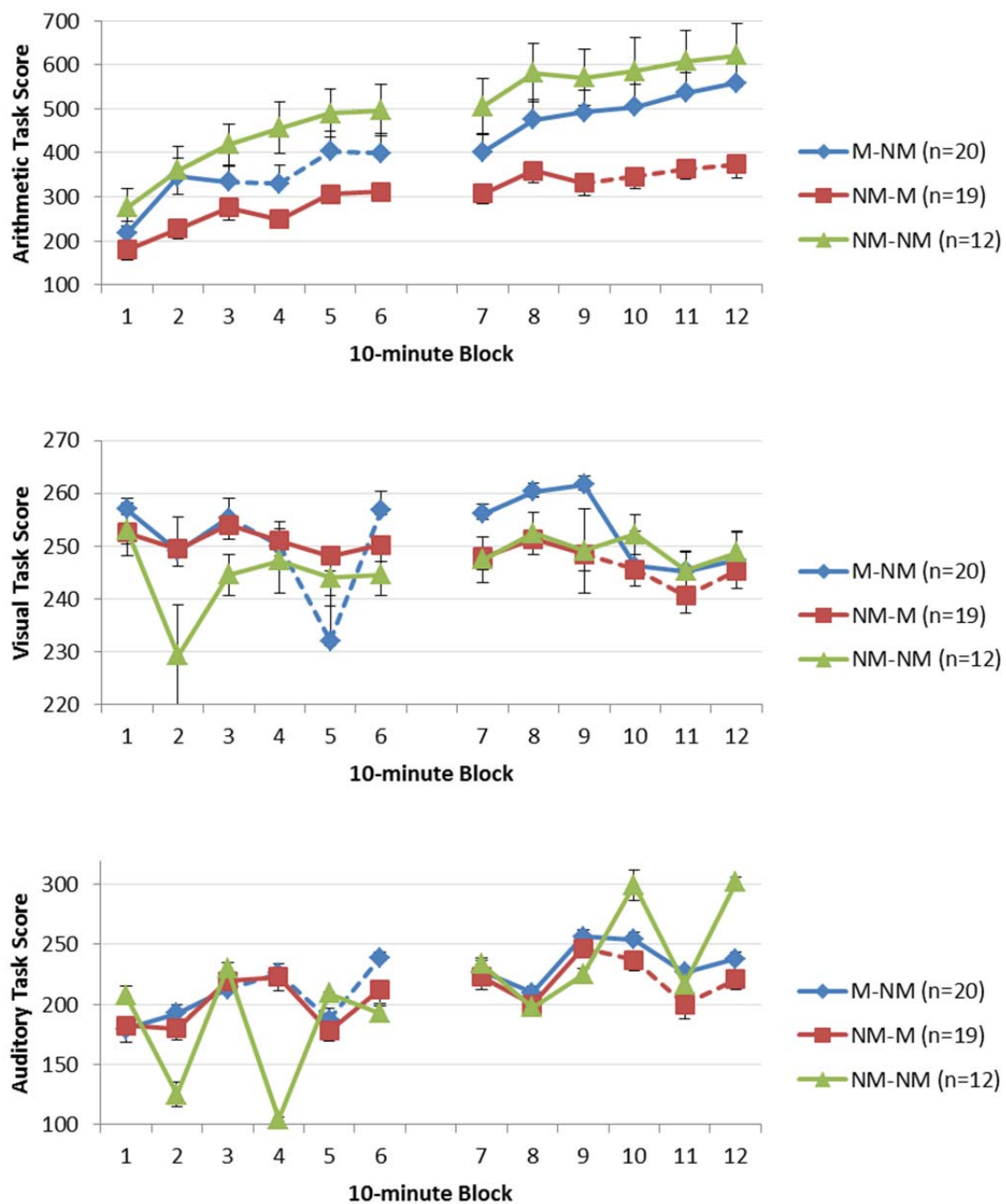


Figure 14. SYNWIN scores per participant group and ten-minute block (absolute values).

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